ANATOMY

- The upper lid and lower lid join laterally at lateral canthus and joins medially is called medial canthus
- Normally lateral canthus is higher up than medial canthus

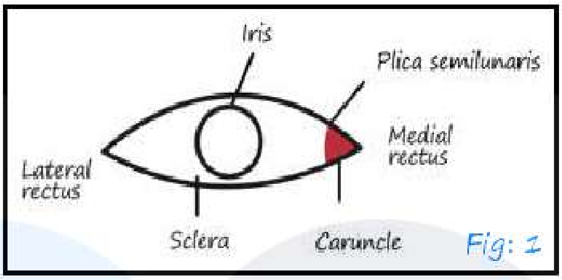

Iris
Plica semilunaris
Medial rectus
Lateral rectus
Sclera
Caruncle

Fig: 1

- Pink area in medial canthus is caruncle

↓

Modified skin

↓

Appendages are present

- Pinkish semilunar band behind caruncle

↓

Plica semilunaris

↓

Vestigial structure in eye

- Colored portion – iris
- Sclera – white
- Aperture in iris is called pupil
- Normal size of pupil- 2-4 mm
- Pupil and iris are covered by transparent layer called cornea
- Sclera is covered by transparent layer conjunctiva

DOC TUTORIALS LOGIN FOR SUCCESS

- Limbus → corneoscleral junction /Corneo conjunctival junction
- Normally size of limbus 1 -2 mm

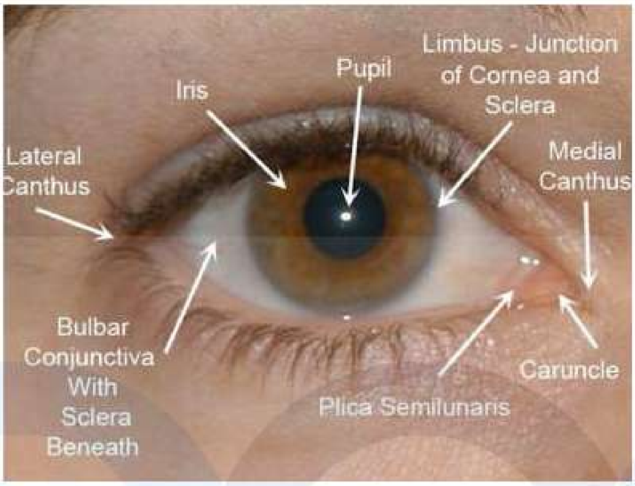

Iris
Pupil
Limbus - Junction of Cornea and Sclera
Lateral Canthus
Medial Canthus
Bulbar Conjunctiva With Sclera Beneath
Plica Semilunaris
Caruncle

Fig: 2

Limbus 00:06:27

- Most important area
- Has stem cells → totipotent cells
  → Capacity of self-renewal
  → Can predispose to disorder
  ↓
  - Dry eye
  - Pterygium
  - Symblepharon
- Limbal specific marker → 'ABCG2'
- Incisions for anterior segment surgeries at level of limbus

Antero-Posterior representation of eyeball

00:08:56

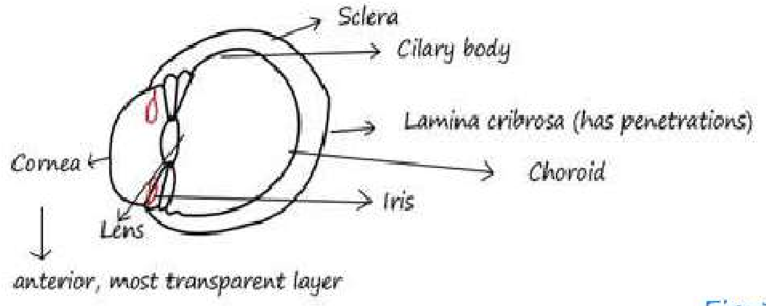

Sclera
Cilary body
Lamina cribrosa (has penetrations)
Cornea
Choroid
Iris
Lens
anterior, most transparent layer

Fig: 3

- Lens is suspended in eye with help of suspensory ligaments /Zonules
- Around 70-80 zonules are present in each eye

Coats of Eye Ball 00:12:32

- Outer most → fibrous → made up of corneal in front and sclera posteriorly
- Middle → vascular → from iris, ciliary body , choroid
- Ciliary body → Anterior → projections → pars plicata

  → Posterior → plain → pars plana
- Pars plicata site of secretion of aqueous humor
- Pars plana – basically a little region , though which you can enter eye for posterior segment surgeries/intravitreal injections
- Intravitreal infections generally given
- 4 mm behind limbus in normophakics
- 3.5 mm behind limbus in psyedophakics
- 3 mm behind limbus in aphakics

DOC TUTORIALS
LOGIN FOR SUCCESS

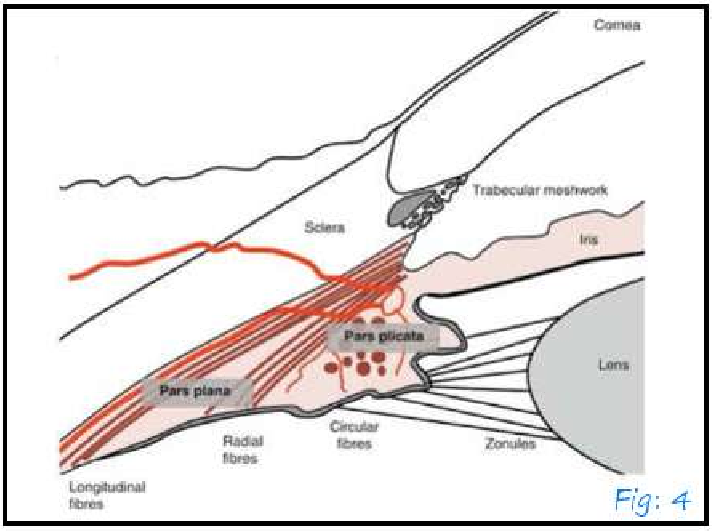

Cornea
Trabecular meshwork
Iris
Sclera
Pars plicata
Lens
Pars plana
Circular fibres
Radial fibres
Zonules
Longitudinal fibres

Fig: 4

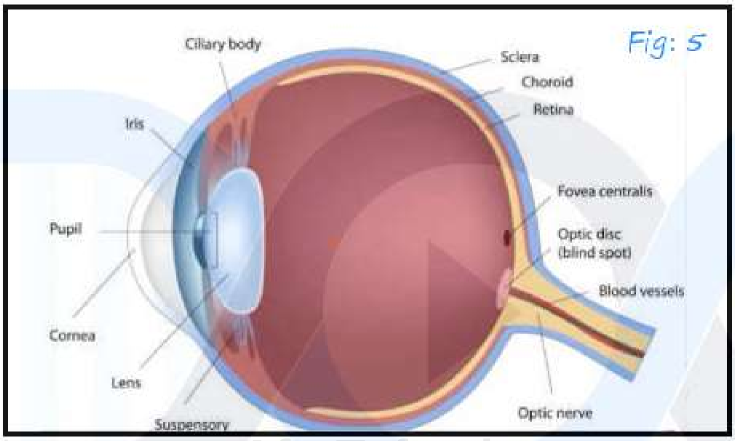

Ciliary body
Sclera
Choroid
Retina
Iris
Fovea centralis
Pupil
Optic disc (blind spot)
Blood vessels
Cornea
Lens
Suspensory
Optic nerve

Fig: 5

- Ciliary body → 6 mm in length
  → has triangular cross section
- Ciliary muscles → 3 types depending on arrangement

i. Circular	ii. Radial	iii. Longitudinal
Responsible for accommodation		Responsible for drainage & Aqueous

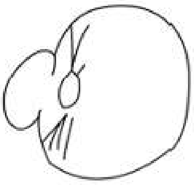
- Innermost → Retina → Neural coat
  ↓
  incomplete coat

Ora serrata – Anterior most part of retina

– Thinnest portion of retina

Segments of Eye Ball 00:21:30

- Divided eye ball in to 2 segments'



Anterior
↓
Has fluid called aqueous humor

Posterior
↓
vitreous humor
↓
4 ml

- Anterior segment divided into 2 chambers

Anterior chamber	Posterior chamber
•Area between cornea and iris	• Area between iris and lens
•Volume: 0.25ml	• Volume 0.06 ml

DOC TUTORIALS LOGIN FOR SUCCESS

- Aqueous humor – Produced throughout life
  - Like a clear solution
- Vitreous humors – Volume is fixed at birth itself
  - Has type II collagen and lot of Hyaluronic acid
  - Like a gel
  - Attached to various structures,

i. Strongest attachment → retina : at base of vitreous near Ora Serrata
ii. Posterior pole
iii. Fovea
iv. Behind the lens

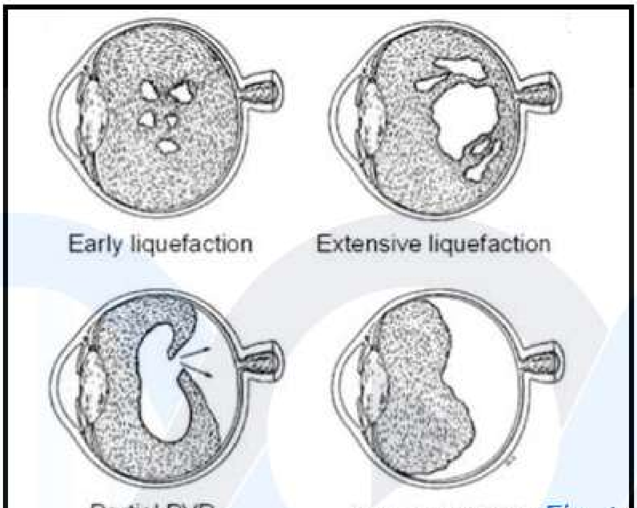

Early liquefaction
Extensive liquefaction
Partial PVD
"Complete" PVD

Fig: 6

- As we grow, vitreous undergoes changes,
- Gel ⟶ liquefied ⟶ contracting
- Mainly occurring at posterior PVD - posterior vitreous detachment
- Sudden PVD→ Phegmato retinal detachment

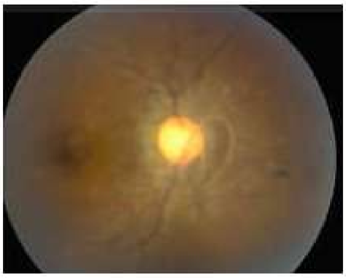
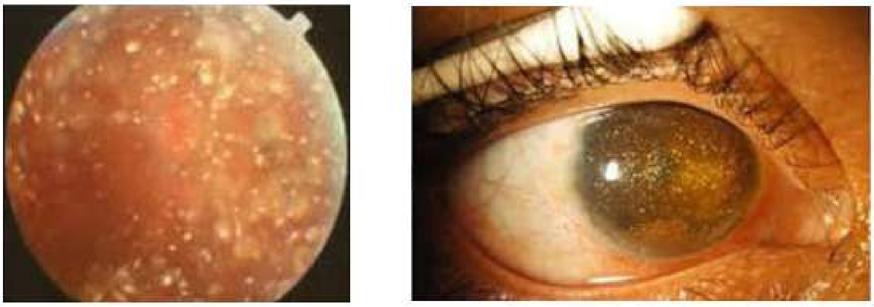


Fig: 7

↓

complete PVD - Weiss ring



Synchisis Synresis

Fig: 8

- Abnormal accumulation in vitreous

↓

Asteroid hyalosis

↓

- Accumulation of calcium in vitreous cavity
- Complains of floaters
- Gel vitreous

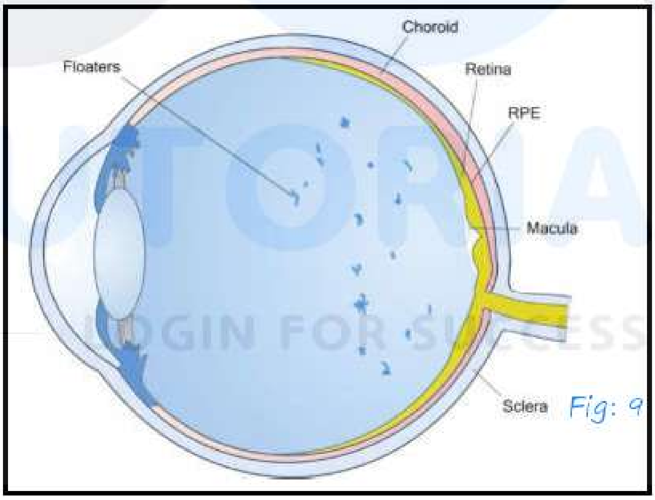

Choroid
Floaters
Retina
RPE
Macula
Sclera

Fig: 9

- Synchesis scintillans
  - Liquefied vitreous
  - Accumulation of cholesterol crystals (Golden brown)

↓

Golden shower

  - complaints of floaters

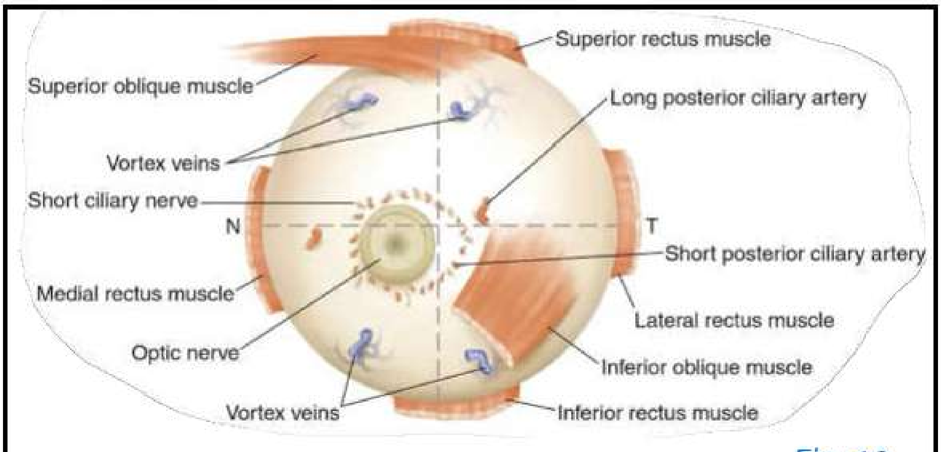

Superior rectus muscle
Superior oblique muscle
Long posterior ciliary artery
Vortex veins
Short ciliary nerve
N
T
Short posterior ciliary artery
Medial rectus muscle
Lateral rectus muscle
Optic nerve
Inferior oblique muscle
Vortex veins
Inferior rectus muscle

Fig: 10

vertex veins

- Vortex vein → drain choroid

→ Mostly 4, sometimes 5-7 vortex veins

↓

@ 2'o clock

5 'o clock

7 'o clock

10 'o clock

AraTafreeh.com

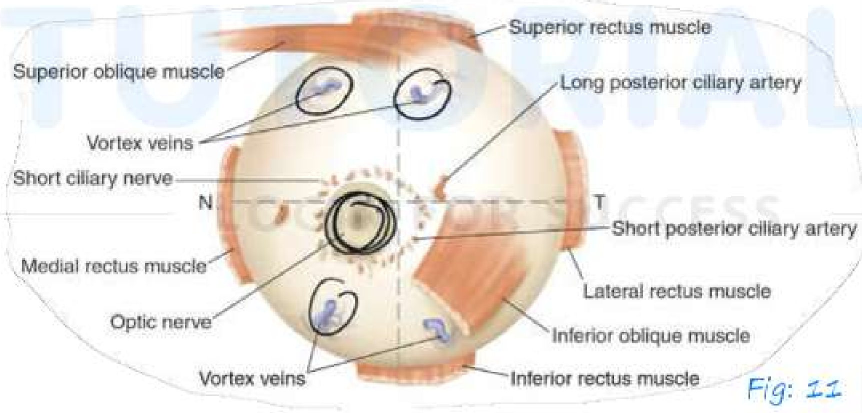

Superior rectus muscle
Superior oblique muscle
Long posterior ciliary artery
Vortex veins
Short ciliary nerve
N
T
Short posterior ciliary artery
Medial rectus muscle
Lateral rectus muscle
Optic nerve
Inferior oblique muscle
Vortex veins
Inferior rectus muscle

Fig: 11

→ Veins of fundus retinal equatorial - imaging area 14 mm behind limbus

- Clinically seen over ampullae of vortex veins

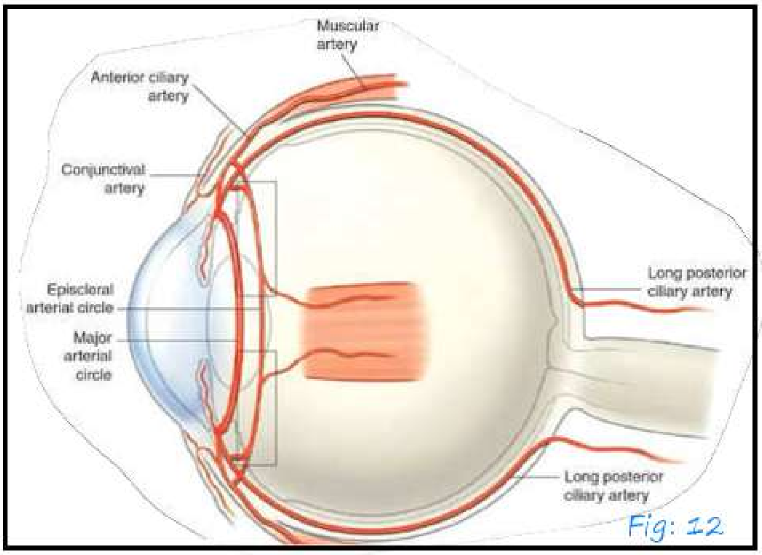

Muscular artery
Anterior ciliary artery
Conjunctival artery
Episcleral arterial circle
Major arterial circle
Long posterior ciliary artery
Long posterior ciliary artery

Fig: 12

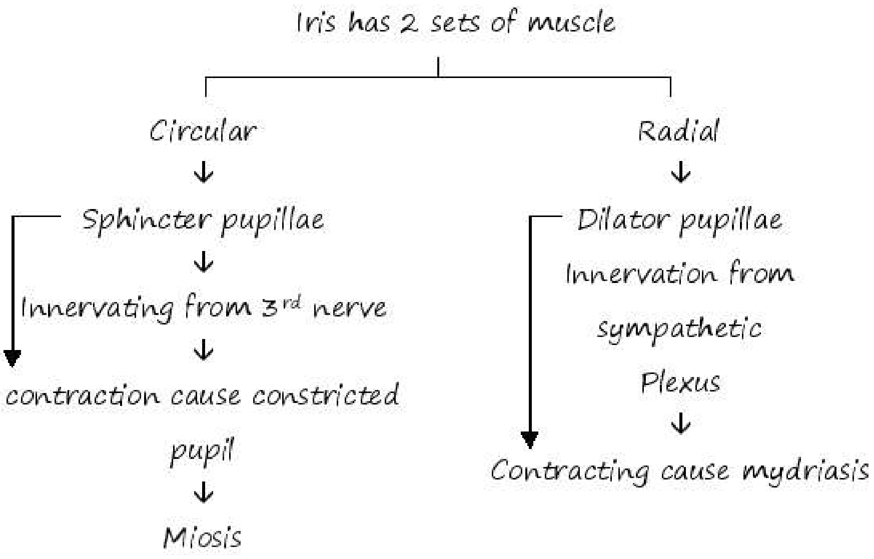
Blood supply of Eye 00:37:09

- Chief artery – ophthalmic artery
- Central retina artery supplies retina

Ophthalmic artery
- Central retinal artery
- Choroidal artery
  - Posterior ciliary artery
    - Short ciliary a
      ↓
      - 20 in number
      - Supply near the optic nerve
    - Long post C.1
      ↓
      - 2 in numbers
      - Supply anterior structures
      - Anterior with anterior ciliary artery the root of iris
        ↓
      - Major arterial circle
        ↓
        Near the root of iris
      - They make their way and makes minor arterial circle @ pupillary margin
  - Muscular
    ↓
    Anterior ciliary

Small Large pupil

TUTORIALS LOGIN FOR SUCCESS


Iris has 2 sets of muscle
Circular
Sphincter pupillae
Innervating from 3rd nerve
contraction cause constricted pupil
Miosis
Radial
Dilator pupillae
Innervation from sympathetic Plexus
Contracting cause mydriasis

Small	Large pupil
• Physiological	• Physiological
- Neonate	- Awake
- Elderly	- Dark room
- Sleeping	- Emotional
- Bright light	- Myopia
• Pathological	• Pathological
- Pilocarpine	- Cycloplegics
- Acute anterior uveitis	- Angle closure glaucoma
- Opioids	- Datura/Belladonna/cocaine
- Op poisoning	- 3rd nerve palsy
- Horners syndrome	- Adie pupil tonic
- Argyl Robertson pupil (B/L small)	- Pontine hemorrhage
- Pin point pupil	

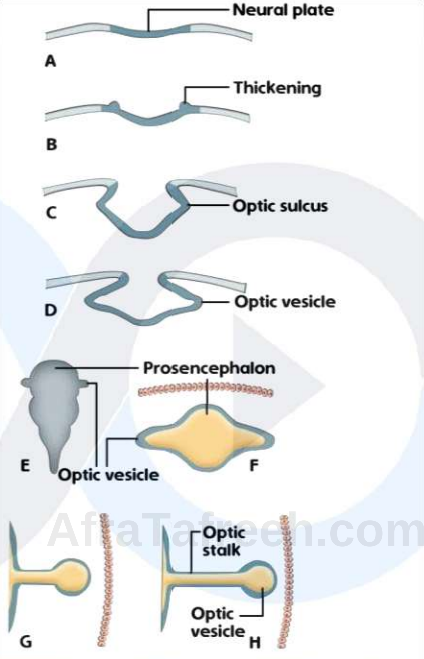
AfraTafreen.com
TUTORIALS
LOGIN FOR SUCCESS

Eyeball Newborn	Adult
• Axial length – 16 mm (A.P diameter) ↓ Measured by USG-A scan	24 mm
• Cornea – 9mm ⟶ 2yrs	11 mm
• Lens 6.5 mm	10 mm
• Accommodation → power by closer objects clearly ↓ Highest	Decreases with age
• Vision → poor ⟶ 5-6 yrs. → < 6/60	6/6 on snellens chart
• Refractive error ↓ Hypermetropia +2 to +3 Doppler	Emmetrope
• Orbit → more divergent orbit	Decreases
• Macula → fully developed	4-6 months after birth
• Lacrimal glands – do not produce tears in 1st few days & birth	

TUTORIALS LOGIN FOR SUCCESS

EMBRYOLOGY

- Eyeball starts developing in 3rd month of gestation (22nd day)
- Master gene for development of eyeball PAX-6 gene.
- Endoderm has no role in eye


Neural plate
A
Thickening
B
Optic sulcus
C
Optic vesicle
D
Prosencephalon
E
Optic vesicle
F
Optic stalk
G
Optic vesicle
H

From diencephalon Fig: 1

DOT TUTORIALS LOGIN FOR SUCCESS
AftaTafreeh.com

- Diencephalon is neuroectoderm
- At 22nd day of gestation – thickening of neuro plate.

↓

Groove – sulcus

↓

Vesicle

- Neuroectoderm Stalk → optic nerve
- Surface ectoderm is also near neuroectoderm

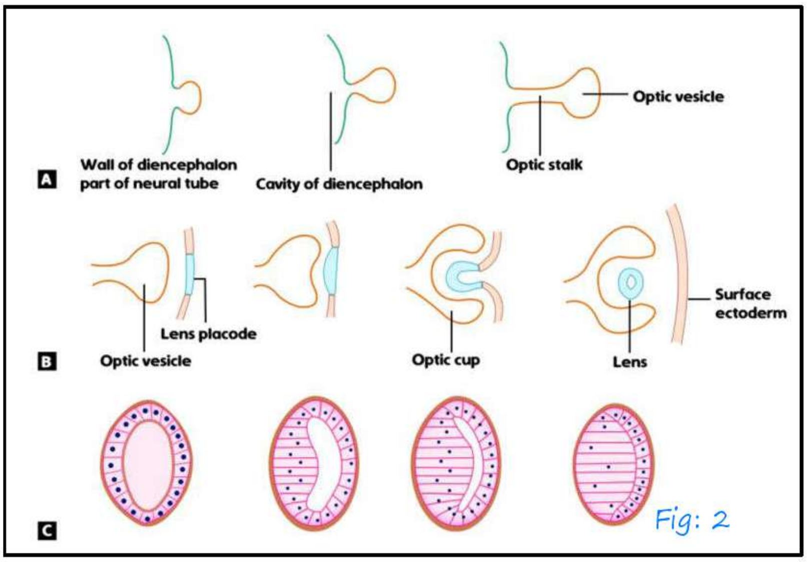

A
Wall of diencephalon part of neural tube
Cavity of diencephalon
Optic stalk
Optic vesicle
B
Optic vesicle
Lens placode
Optic cup
Lens
Surface ectoderm
C

Fig: 2

- From Vesicle → optic cup
- Surface ectoderm → thickening (lens placode)

↓

Invagination in the cup and finally detaches from surface ectoderm

↓

Lens vesicle

↓

Future lens

Neuroectoderm 00:06:28

ROMES

- Retina
- Optic nerve
- Muscle of iris
- Epithelium of iris, ciliary body
- Secondary vitreous

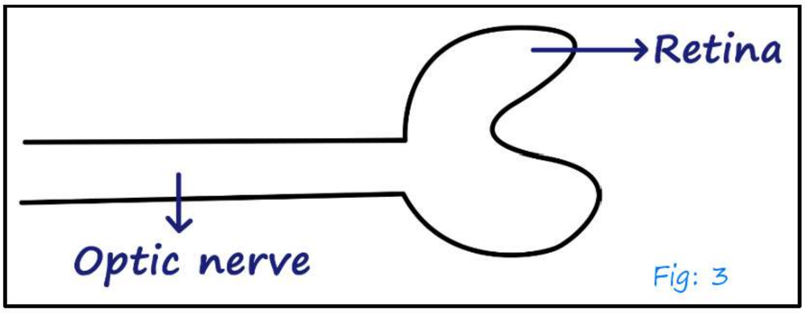


Fig: 3

- Outer layer of cup
  - Retinal pigment epithelium
  - Epithelium of ciliary body and epithelium of choroid
- Inner layer of cup forms neuro sensory rretina.

Surface Ectoderm 00:09:13

- L-Lens, Lacrimal gland, lid
- E – Epithelium of conjunctiva and cornea
- G – Glands

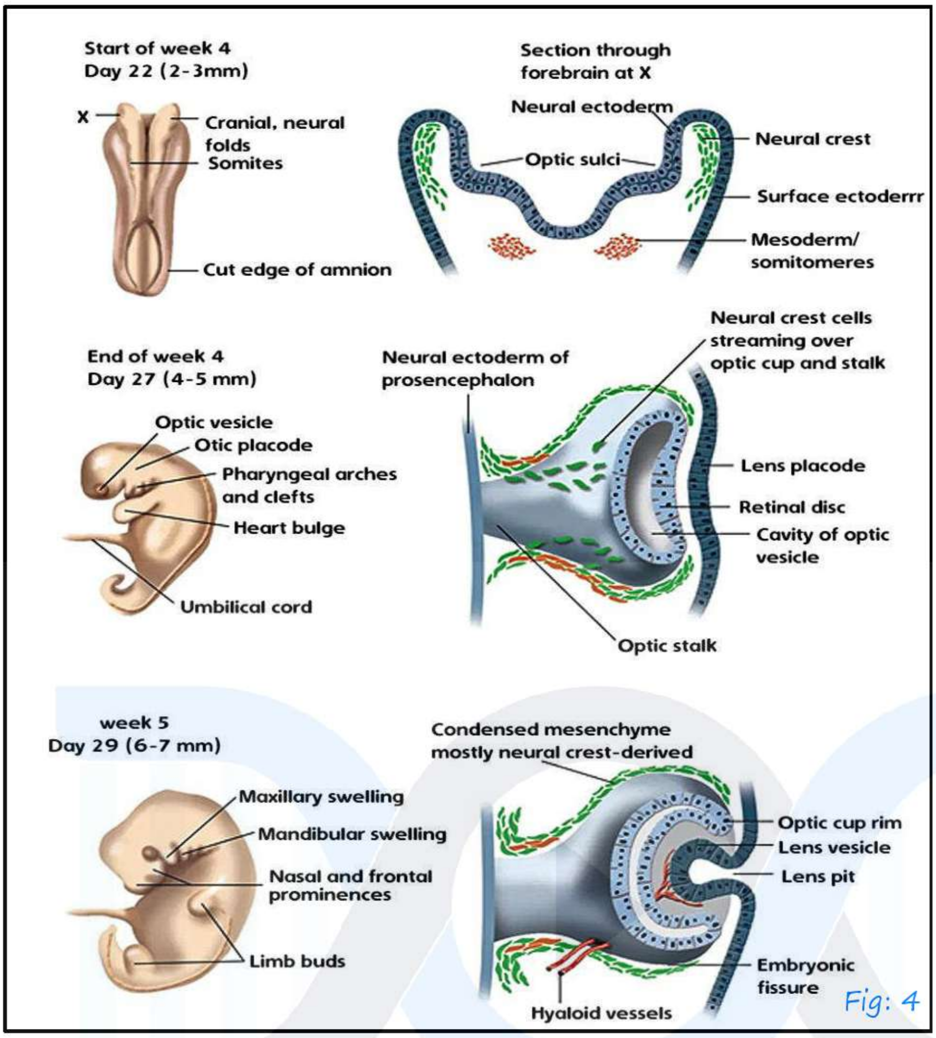

Start of week 4
Day 22 (2-3mm)
X
Cranial, neural folds
Somites
Cut edge of amnion
Section through forebrain at X
Neural ectoderm
Optic sulci
Neural crest
Surface ectoderrr
Mesoderm/ somitomeres
End of week 4
Day 27 (4-5 mm)
Optic vesicle
Otic placode
Pharyngeal arches and clefts
Heart bulge
Umbilical cord
Neural ectoderm of prosencephalon
Neural crest cells streaming over optic cup and stalk
Lens placode
Retinal disc
Cavity of optic vesicle
Optic stalk
week 5
Day 29 (6-7 mm)
Maxillary swelling
Mandibular swelling
Nasal and frontal prominences
Limb buds
Condensed mesenchyme mostly neural crest-derived
Optic cup rim
Lens vesicle
Lens pit
Embryonic fissure
Hyaloid vessels

Fig: 4

- Neural crest cells
  - Develop from ectoderm
  - Special cells
  - Generally migrate, between surface ectoderm and rest on neural ectoderm

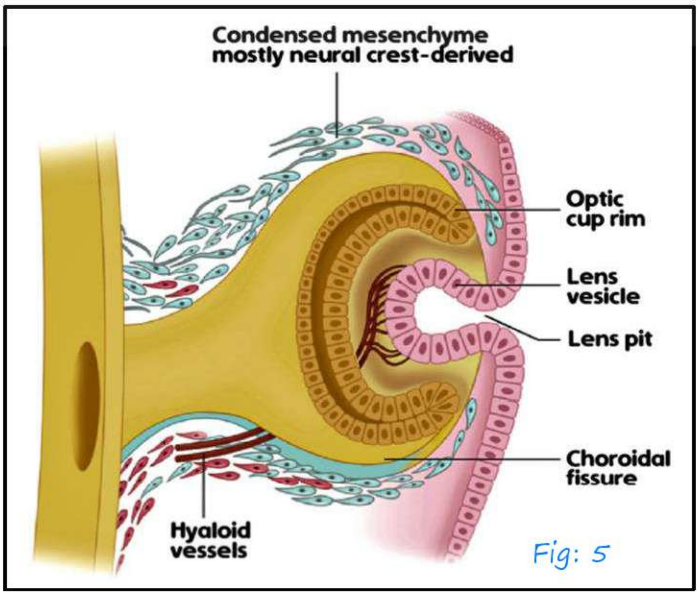

Condensed mesenchyme mostly neural crest-derived
Optic cup rim
Lens vesicle
Lens pit
Choroidal fissure
Hyaloid vessels

Fig: 5

- Mesenchyme = Mesoderm + Neural crest

Neural Crest 00:12:26

Neural

- Ciliary ganglion
- Optic nerve covering
- Myelin sheath (oligodendrites)
- Melanocytes

Mesenchymal → cost

- Cornea except epithelium, ciliary muscle, choroid
- Orbital bones
- Sclera except temporal part, Schlemm canal, stroma of iris, ciliary body TM

Mesoderm 00:15:25

PEST

1. Primary vitreous
2. Endothelium of blood vessels, Extra ocular Muscles
3. Sclera (Temporal part)

Vitreous 00:17:01

- 2° vitreous → adult → neuroectodermal origin
- 1° vitreous → during gestation → mesodermal origin
- 3° vitreous → zonules

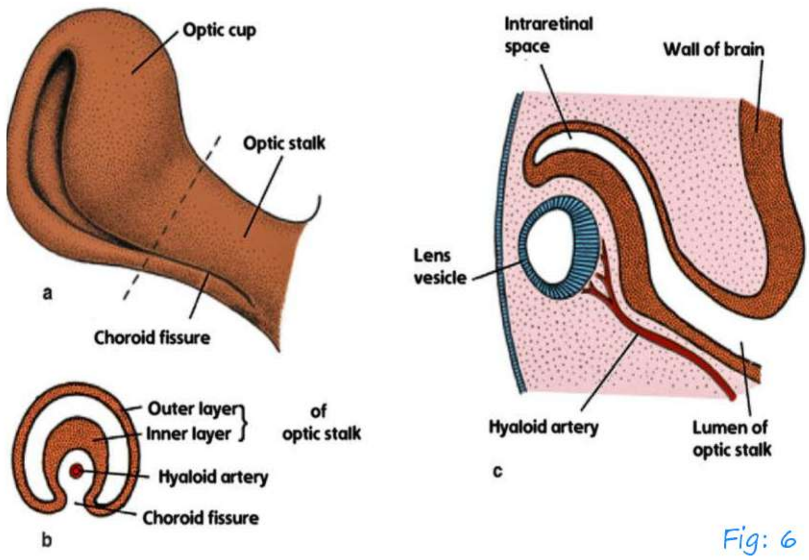

Optic cup
Optic stalk
a
Choroid fissure
Outer layer
Inner layer
of optic stalk
Hyaloid artery
Choroid fissure
b
Intraretinal space
Wall of brain
Lens vesicle
Hyaloid artery
Lumen of optic stalk
c

Fig: 6

Nourishnment through hyaloid artery to the lens

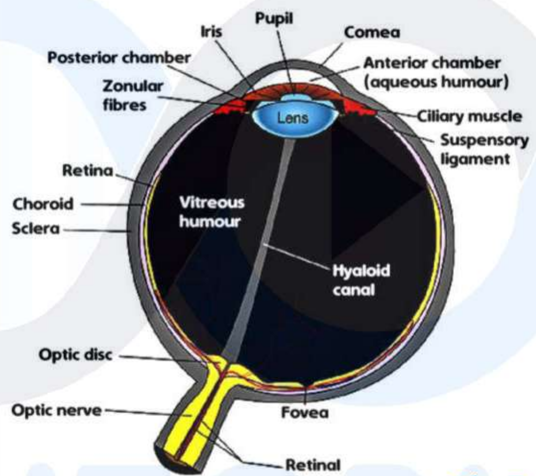

Pupil
Iris
Cornea
Posterior chamber
Anterior chamber (aqueous humour)
Zonular fibres
Lens
Ciliary muscle
Suspensory ligament
Retina
Choroid
Sclera
Vitreous humour
Hyaloid canal
Optic disc
Optic nerve
Fovea
Retinal blood vessels

Fig: 7

- Hyaloid canal/cloquet's canal → transport remanent – when hyaloid artery disappear it leaves it in vitreous cavity.
- Hyaloid artery retracts and forms CRA at back.
- Hyloid vessels forms 1° vitreous
- Hyaloid fissure in inferior part of neuroectodermal cup

  ↓

  Hyaloid vessels give nourishment to developing lens

- Fissure disappears or closes at 6-7th week of gestation.

↓

Hyaloid vessels retracts → proximal part

↓

Central retinal artery

1° vitreous ← transparent hyaloid canal ← distant part disappears

- If fissure does not close → IRIS COLOMBOMA

↓

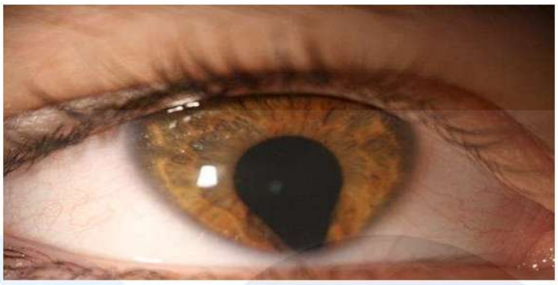


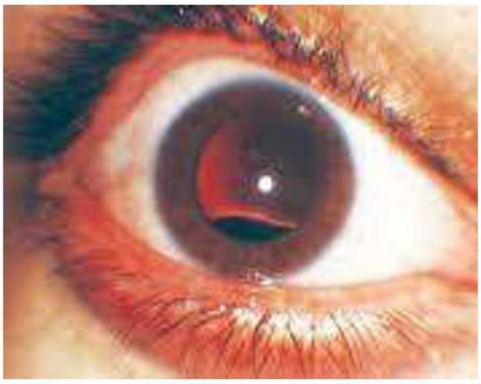
Fig: 8

- Word 'coloboma' – defected
  - mutilated
  - key hole iris
- Based on location of coloboma

Typical	Atypical
↓	↓
Inferonasal	Anywhere else

- Choroidal colomba

Zonular coloboma



Lens coloboma (Zonular) Fig: 9

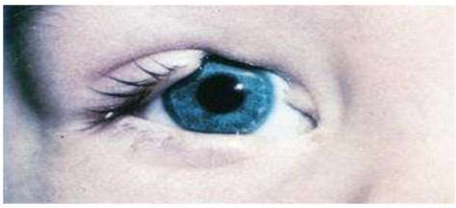


Fig: 10

↓

Lid coloboma

↓

- Type of facial cleft
- Can be in upper lid or lower lid

↓ Medial part of lid

↓ Lateral part of lid

- What will happen if 1° vitreous/hyaloid artery does not disappear

↓

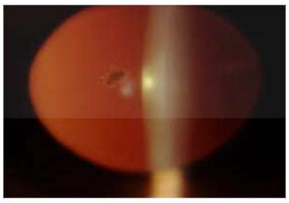
PHPV → Persistent hyperplastic primary vitreous

Anterior

↓

Behind lens

↓



Mittendorf dot

↓

Small opacity at posterior part of lens

Posterior

↓

Near optic nerve head

↓

Bergmeister papillae

Tunica vasculosa lentis 00:29:28

- Capillary network that branches from the hyaloid artery and covers the lens surface.
- Anteriorly, it has attachments that extend to the pupillary frill of the iris.
- Posteriorly, anastomosis with the hyaloid system and is attached to the ciliary process.

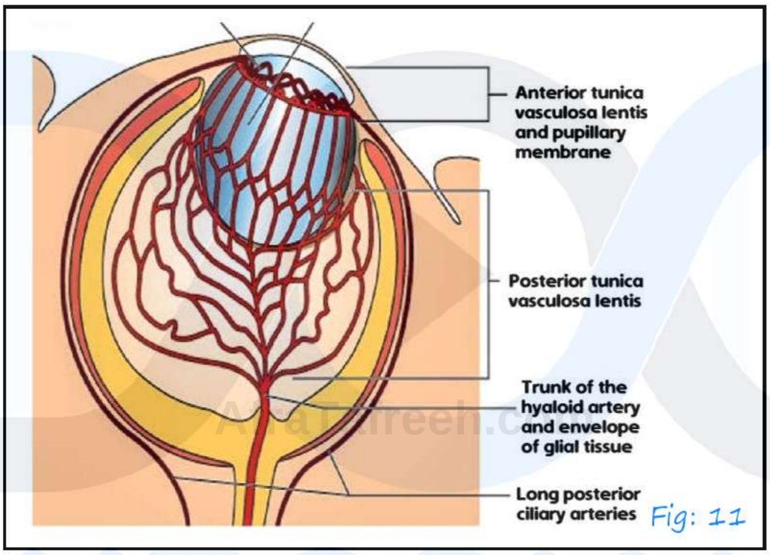

Anterior tunica vasculosa lentis and pupillary membrane
Posterior tunica vasculosa lentis
Trunk of the hyaloid artery and envelope of glial tissue
Long posterior ciliary arteries

Fig: 11

Persistant pupillary membrane 00:30:00

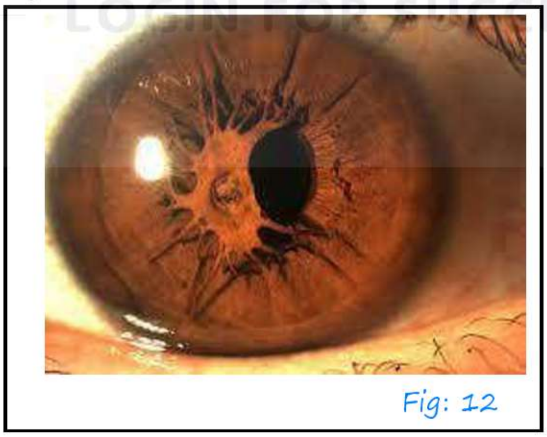


Fig: 12

- Congenital anomaly
- Persistance of remanents of hyaloid artery or tunica vasculosa testis

Retina 00:30:55

- Inner wall differentiates into 3 layers

  1. Matrix – forms rods & cones
  2. Mantle – forms bipolar cells and ganglionic cells as well as other neurons of retina
  3. Marginal – forms optic nerve axons of ganglion cells from marginal layer that converges towards optic stalk and finally farms optic nerve.

Neural Crest wave 00:31:21

- Starts in 7th week
  - 1st wave – corneal and trabecular endothelium
  - 2nd wave – formation of stroma of cornea
  - 3rd wave – formation of iris stroma

Eyelid 00:32:00

- 1st month lid develops
- Lid fuses 3rd month
- Retracts in 5th month
- Ablepharon = absence of eyelid fold
- Ablepharon macrostomia syndrome

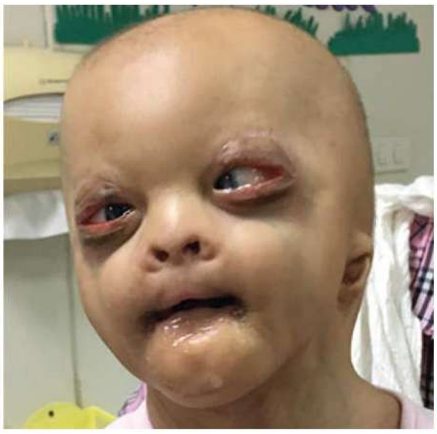


Fig: 13

- Cryptophthalmos – if eyelid fails to separate
- Ankyloblepharon:

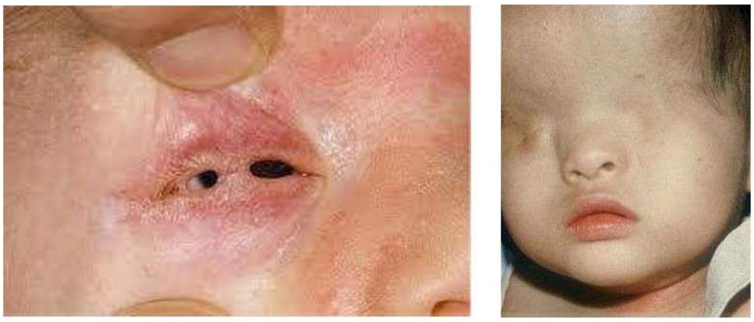


Fig: 14

- Ankyloblepharon – adhesion between lower and upper lid.

Clinical importance

- PAX 6 gene
  - Anophthalmia- absence of eyeball
  - Microphthamia – length <19 mm at 1 year or <21mm in adult

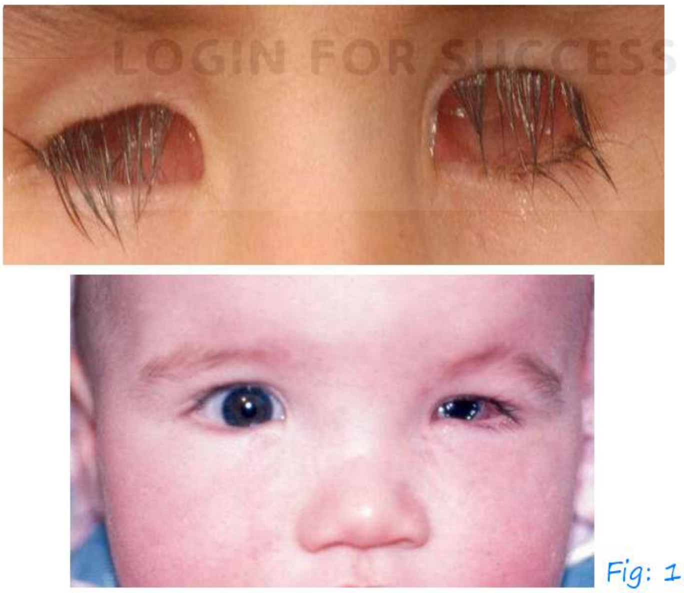


Fig: 15

- Nanophthalmos → normal small eye
- Micropthalmia – small abnormal eye
- SHH gene – (sonic hedgehog gene) for development of both eye
- Cyclopia

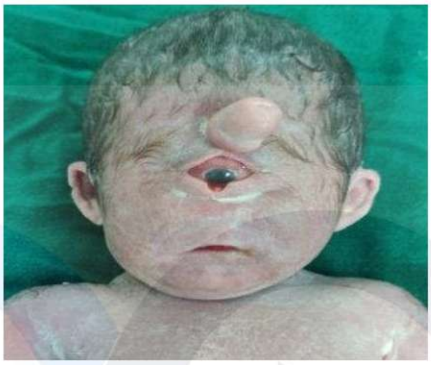
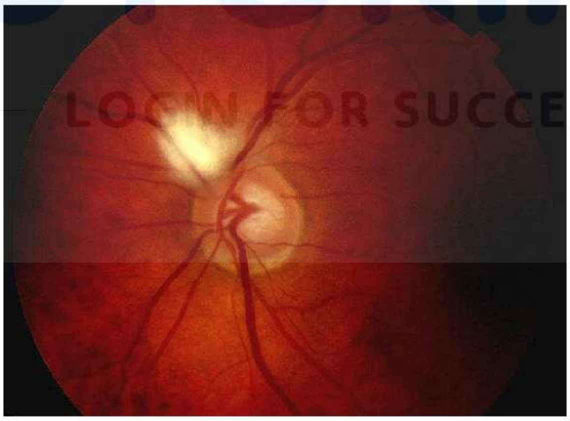


Fig: 16

- Mutation in SHH gene → cyclopia → single centre eye.
- Axenfeld – Rieger syndrome is a rare genetic condition that may be due to defects affecting the optic cup.
- Mutation associated with ARS is in the transcription factor PITX2.
- Morning glory syndrome – merodermal defect in development of sclera

Developmental time	Ocular development
3RD WEEK	OPTIC GROOVE FORMS
4 WEEKS	LENS VESICLE FORMS , HYALOID VESSELS
6 WEEK	CLOSURE OF EMBRYONIC FISSURE
4TH MONTH	HYALOID VESSELS REGRESS, RETINAL VESSELS BEGIN TO FORM
6TH MONTH	NLD BECOMES PATENT
7TH MONTH	MYELINATION BEGINS
8TH MONTH	HYALOID VESSELS DISAPPEAR



Myelinated nerve fibres Fig: 17

Age	Visual milestone
At birth	Wandering/ momentary fixation
6-8 weeks	Fixation completed
3 months	Follows movements , binocular vision
5-6 months	Depth, color perception Macula fully developed

ANATOMY, LAYERS, LYMPHATIC DRAINAGE

Conjunctiva 00:00:08

Parts of conjunctiva:

- Bulbar conjunctiva: Over sclera
- Palpebral conjunctiva: Behind eye lid
- Fornix: It is a sac between bulbar and palpebral conjunctiva

  Word conjunctiva is derived from word conjoin – Meaning to joint

  ↓

  It joins eye ball with eye lid.

Palpebral conjunctiva

- Marginal conjunctiva – 2mm
- Tarsal conjunctiva
- Orbital conjunctiva

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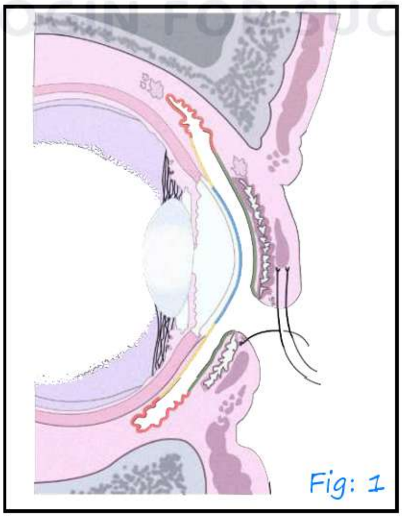

Fig: 1

Visualization of upper fornix: -

- It is visualized using Desmarre's lid retractor
- The method used is double eversion method
- It is used in various lid surgeries
- Upper fornix is most common site for foreign body lodgment and chemical injury

Glands in conjunctiva 00:06:42

- Mucin secreting glands
  1.Goblet cells:
    - Mucin producing glands
    - Mucin forms inner most layer of tear film
    - Goblet cells, maximum in nasal part of bulbar conjunctiva
  2.Gland of Man$_2$
  3.Glands / crypts of Henle
- Accessory lacrimal glands:
  - Gland of Krause
  - Gland of wolfring

Location of glands

Gland	Location	Mnemonic
MANZ	At limbus	(MAN – on his limbs
Krause	At fornix	(krow-flying)
Henle	Tarsal	(Hunter)
wolfring	Palpebral conjunctiva	(Ring palpate)

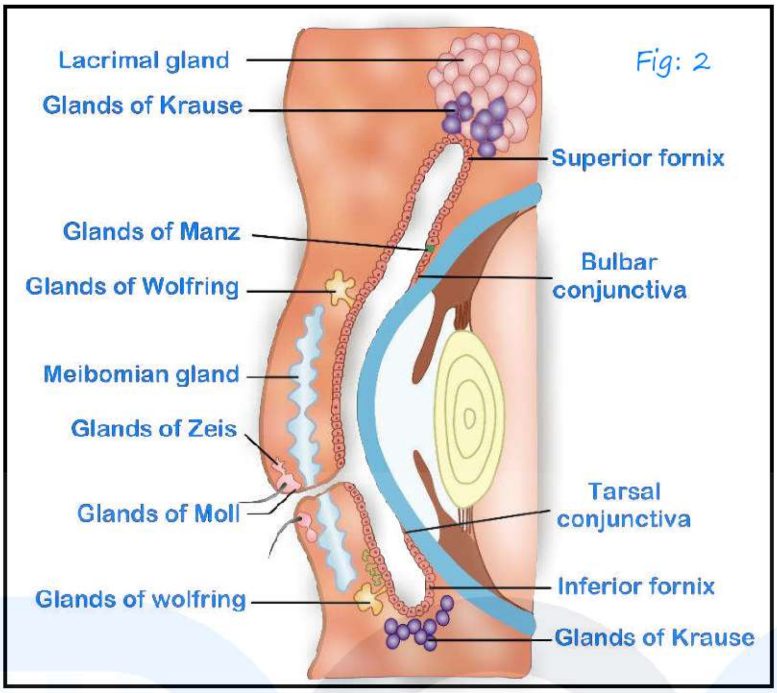
Lacrimal gland
Glands of Krause
Superior fornix
Glands of Manz
Glands of Wolfring
Bulbar conjunctiva
Meibomian gland
Glands of Zeis
Glands of Moll
Tarsal conjunctiva
Inferior fornix
Glands of wolfring
Glands of Krause

Fig: 2

Layers of conjunctiva 00:11:18

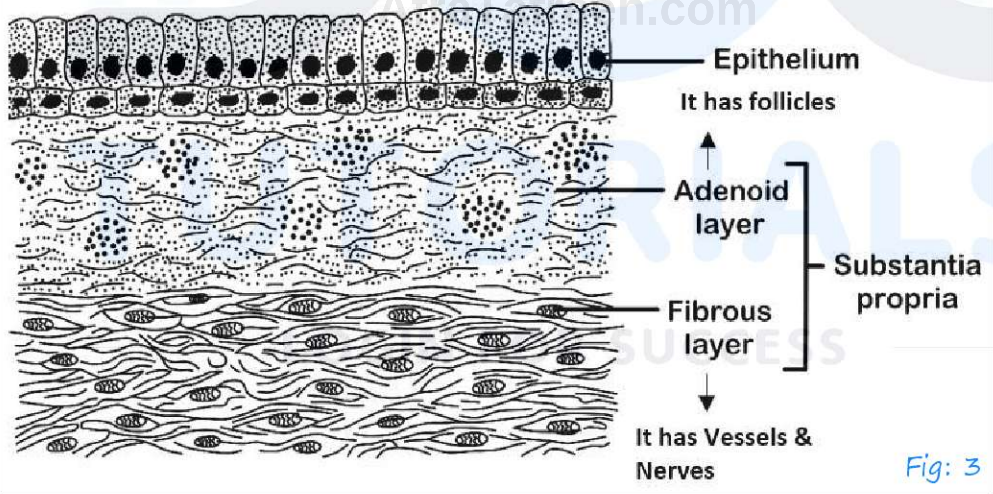
Epithelium
It has follicles
Adenoid layer
Substantia propria
Fibrous layer
It has Vessels & Nerves

Fig: 3

Note:

Follicles are not very developed till 3 months of age

Therefore, follicular conjunctivitis never there in neonates

Lymphatic drainage in conjunctiva 00:13:30

- Only structure of the eye to have lymphatic drainage in conjunctiva

  Medial part of conjunctiva: Submandibular lymph nodes

  Lateral part of conjunctiva: Preauricular and parotid nodes.

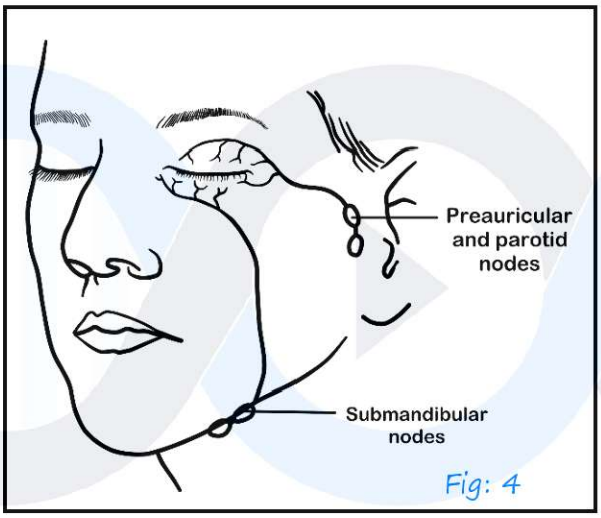
Preauricular and parotid nodes
Submandibular nodes

Fig: 4

CONJUNCTIVITIS

- Inflammation of conjunctiva
- Most common type is viral (most commonly adenovirus)

Symptoms

- Redness
- Foreign body sensation
- Itching
- Watering
- Discharge: Colored halos: seen in acute mucopurulent conjunctivitis due to splitting of light by discharge. No pain and no loss of vision in conjunctivitis

Redness

	Conjunctival congestion	Circum corneal congestion
Area involved	Superficial conjunctivitis - posterior conjunctival artery involved	Deep conjunctivitis - anterior ciliary vessels involved
Color	Bright red	Dull red
Position	Towards fornix	Maximum towards cornea
Blanching on pressure /	Blanch ⊕	No blanching

Adrenaline / Phenylephrine		
When squeezed	Fills from fornix to center	From limbus to periphery Eg: - Uveitis - Corneal ulcer - Endophthalmitis - Ange closure glaucoma

Types of Conjunctivitis 00:07:48

1.Membranous conjunctivitis Vs Pseudomembranous conjunctivitis

Membranous Conjunctivitis	**Pseudomembranous Conjunctivitis**
• Exudation over palpebral conjunctiva • Necrosis ⊕ • Not be peeled easily bleed ⊕ Eg: corynebacterium diphtheria, streptococcus hemolyticus, adenovirus	• Exudation present over palpebral conjunctiva • Necrosis absent wiped easily bleed negative Eg: Allergies, viral

2.Angular conjunctivitis

- Confined to angles (Lateral canthus) – Maceration / excoriation occurs due to release of proteolytic enzymes caused by Moraxella axenfeld / lacunata
- It is also known as Diplo bacillary conjunctivitis
- Mostly inoculated from nasal cavity

Treatment: 1% tetracycline ointment BD x 14 days
Zinc oxide ointment at night time

3.Acute mucopurulent conjunctivitis

Most common bacterial conjunctivitis
↓↓
Staph aureus

Colored Halos ⊕ / –

4.Hyperacute conjunctivitis

- It is fulminant infection, also known as: Blennorrhea
- Causative: Neisseria gonorrhoea
  - Occulogenital spread
  - Discharge ⊕
  - Keratitis present
  - Uveitis
  - Perforation of cornea can also occur
  - Various lid complications also seen
  - Lymph node enlargement also present
  - Swelling of bulbar conjunctiva

Treatment of bacterial conjunctivitis 00:17:12

1. Topical antibiotics
2. Tear supplements – soothing to the eye
3. Anti histaminics – Decrease congestion

- In viral conjunctivitis no need of treatment, tear supplements + Anti histaminics can be given for symptomatic relief
- Topical antibiotics can be used as prophylaxis in viral conjunctivitis

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Viral conjunctivitis

Most common type of conjunctivitis

Most common – Adenovirus

Types

1. Viral conjunctivitis with serous discharge
2. Acute follicular conjunctivitis

Follicles present:

↓

- Central lymphoid aggregates surrounded by blood vessels
- Involve adenoid layer
- Causative: Adenovirus Species 1-11, 19

3. Epidemic kerato conjunctivitis

Epidemic ↓ Fast spreading

kerato conjunctivitis ↓ involve cornea

- Caused by adeno virus species 8,19,37
- It is mainly follicular type of conjunctivitis

  - Photophobia is also seen
  - Treatment: Topical steroids +/-

4. Pharyngoconjunctival fever
   - Pharyngitis present
   - Fever present
   - Follicular type of conjunctivitis
   - Causative: Adenovirus 3,4,7

5. Acute hemorrhagic conjunctivitis

Causative [Mnemonic PACE] : Picorna → Enterovirus → Coxsackie → Echovirus

P – Picorna virus

A – Adenovirus – 11

C – Coxsackie 24

E – Enterovirus 70, Echovirus

Most specific for hemorrhagic conjunctivitis – Enterovirus 70

↓

Apollo virus conjunctivitis

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Sub conjunctival Hemorrhage 00:27:30

Differential Diagnosis:

- Trauma- most common
- Hypertension: most common cause in old age
- Blood disorder
- Blood thinners (aspirin)
- Coughing
- Straining at stools
- Whooping cough

- Menses (Vicarious menstrual cycle)

Ligneous conjunctivitis 00:29:30

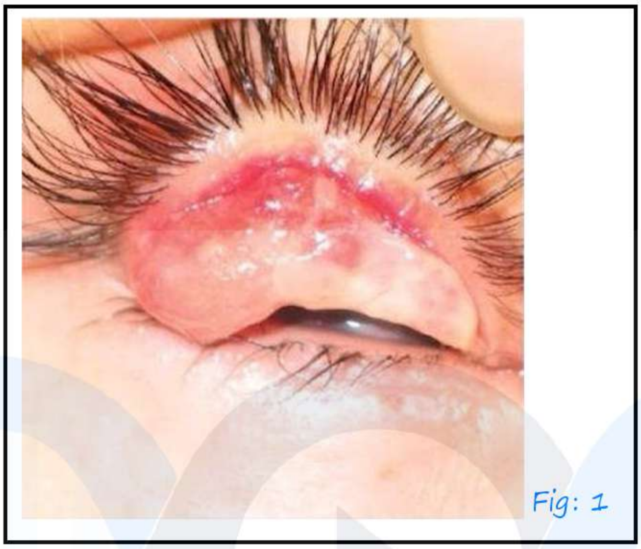

Fig: 1

- Woody and nodular appearance of palpebral conjunctivitis
- Due to mutation in plasminogen gene
- Repeated, frequent mechanical trauma

↓

Healing problem

↓

Abnormal fibrin matrix deposition

TRACHOMA & OPHTHALMIA NEONATORUM

- Common blinding disorder
- One of the most common causes of blindness worldwide.
- Most common infections cause of blindness (worldwide)
- Definition: - It is the chronic inflammation of conjunctiva, and cornea; caused by C. trachomatis type A, B, Ba, and C
- C. Trachomatis serovars: -
  - A, B, Ba, and C → Trachoma
  - D-K
    - Ophthalmia neonatorum
    - Swimming pool blindness (also known as Para trachoma)
    - L1, L2, L3 → Lymphogranuloma venerium
- Acute infection occurs in children
- Chronic infection occurs in elderly

  Source of infection – 3 'F'
  - Fingers
  - Flies
  - Fomites

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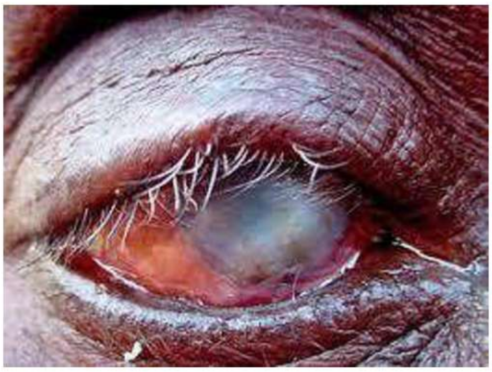


Fig: 1

- C. Trachomatis cycle

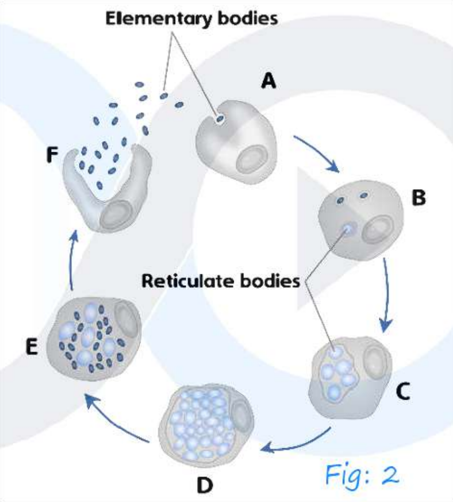
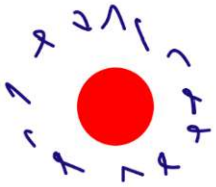

Elementary bodies
A
B
C
D
E
F
Reticulate bodies

Fig: 2

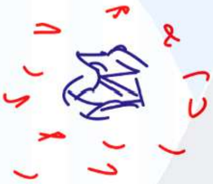
- Elementary bodies – infectious extracellular (But do not replicate)
- Reticulate bodies – Replicating – non infections

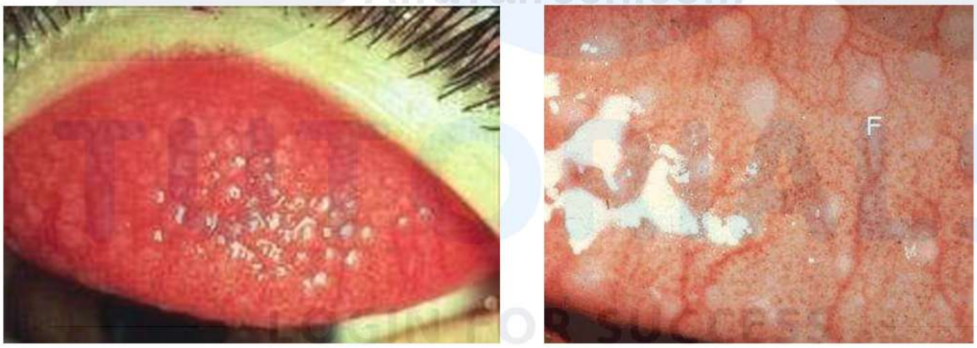
Acute inflammation in Trachoma 00:06:00

- Mainly in children
  - Papillae
- Collection of blood vessels as a central core which are surrounded by aggregation of inflammatory cells
- Velvety appearance



- Follicle
  - Central lymphoid aggregate surrounded by septae of blood vessels
  - Sago grain appearance



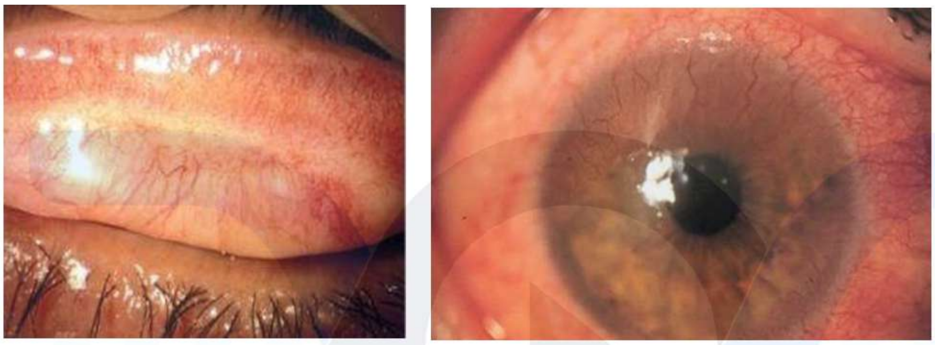


Papillae Follicles Fig: 3

- Follicles may be there in other types of conjunctivitis also but trachomatous follicles can have:
  - Necrosis
  - Leber cells

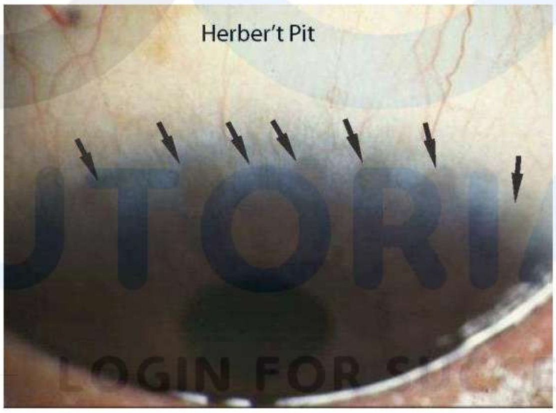
Chronic inflammation 00:08:36

- Aret's line – line of scarring on upper palpebral conjunctiva – At sulcus subtarsalis
- Herbert's pit – scarred, healed follicles over the limbus – They are pathognomonic lesions of trachoma
- Pannus – infiltration + vascularization over cornea



Arets Line

Pannus



Herber't Pit

Fig: 4

Classification of trachoma (By WHO) 00:11:50

- F I S T O – Mnemonic

Classification	Identifying Features
Trachomatous inflammation (follicular) TF	5 or more follicles of >0.5 mm on the upper tarsal conjunctiva
Trachomatous inflammation (intense) TI	Papillary hypertrophy and inflammatory thickening of the upper tarsal conjunctiva, obscuring more than 50% of the deep tarsal vessels
Trachomatous conjunctival (TS)	Grossly visible scars in the tarsal conjunctiva
Trachomatous trichiasis (TT)	1 or more ingrown eyelashes touching the globe, or evidence of epilation (eyelash removal)
Corneal opacity (CO)	Presence of corneal opacity blurring part of the pupil margin.

- Trichiasis – Inturned eye lashes

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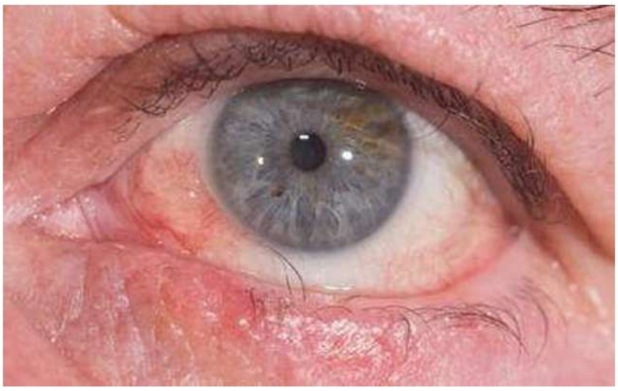

Fig: 5

Sequelae 00:14:15

- Trichiatic cilia
- Eye lash abnormality
- Entropion
- Tylosis – Thickening of lid margin
- Ectropion

Complication: Only one → Ulcer in cornea

Management of Trachoma 00:15:25

S A F E Strategy

- S – Surgery
- A – Antibiotic
- F – Facial cleanliness
- E – Environmental Sanitation

- Surgery: - It is done if the patient has trichiatic cilia (Indication)

  - Bilamellar Tarsal Rotation

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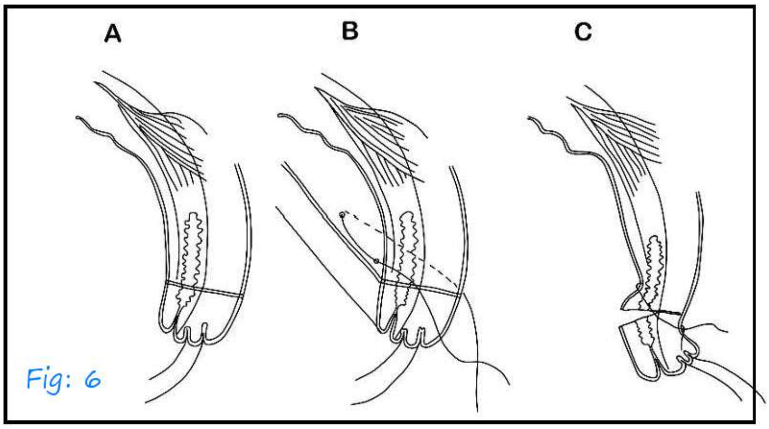


Fig: 6

- Antibiotics: -

  DOC → Azithromycin

  Adult dose → 1gm stat.

  Ideal dose → 20 mg/kg
- If prevalence of trachoma in children 1 to 9 years of age >10% then mass treatment should be given. i.e., treat everyone in the community.
- If prevalence of trachoma in children 1 to 9 years of age is 5-10% then targeted treatment is given i.e., only the respective family is treated
- If prevalence of trachoma in children 1 to 9 years of age is <5%
  - Facial cleanliness
  - Environmental sanitation
- In December 2017 under GET – 2020 program – India is declared Trachoma free

Blanket treatment/ mass prophylaxis :

- In hyperendemic areas where prevalence is >10% use 1% Tetracycline BD x 5 days and continue for 6 months → Intermittent treatment.

Ophthalmia Neonatorum 00:21:20

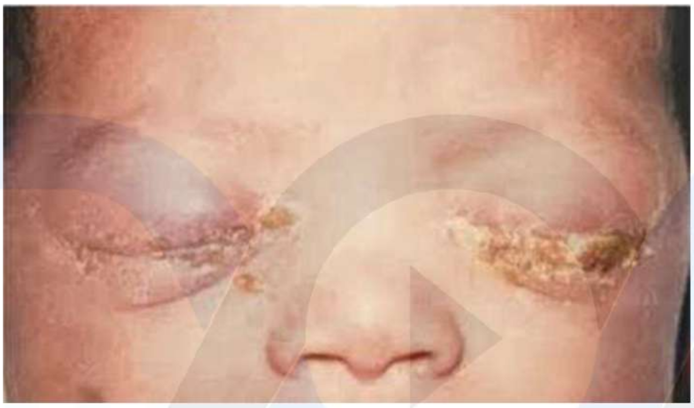


Fig: 7

- Caused by chlamydia trachomatis D-K
- Normally in first few days of life there is no production of tears because lacrimal gland is not fully functional. If at all their tears associated with discharge in first few days of life it is pathological

3 causes of watering in new born: -

1. Ophthalmia neonatorum
2. Congenital glaucoma
3. Congenital nasolacrimal duct obstruction

Other causes of ophthalmia neonatorum:

Mnemonic – Come Girl Out Have Coffee

- Chemical – Credes method: 1 % silver nitrate
  ↓
  Now a days it is not being done

  Now – Erythromycin ointment is given for prophylaxis if required .
- It is very initially after birth i.e., 6 hours or one day within birth
- Gonococcal – After 1-2 days
  - Through maternal genital tract
  - Treat baby, mother, partner
  - Lot of purulent discharge
  - There can be hyperacute conjunctivitis also
  - Most severe form
- Others – after 3-4 days
  - Herpes – after 4 days
  - Chlamydia – after 7 days – most common cause.

ALLERGIC CONJUNCTIVITIS

Allergic conjunctivitis

1. Vernal keratoconjunctivitis
2. Atopic keratoconjunctivitis
3. Gaint cell papillary
4. Phlyctenular conjunctivitis

Vernal keratoconjunctivitis (VKC) 00:00:42

- Type I hypersensitivity (mainly) – IgE.

  Mediated to exogenous allergen

  Type IV may also be seen

  Spring; summer most common
- Also known as spring catarrh
- Also known as warm weather conjunctivitis

Clinical features 00:02:22

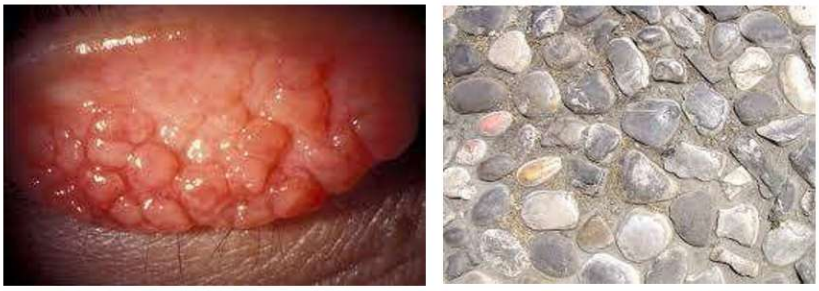
- More common in male children (1st decade)
- frequent itching – summer
- Signs
  - Papillary response – cobble stone appearance

    ↓

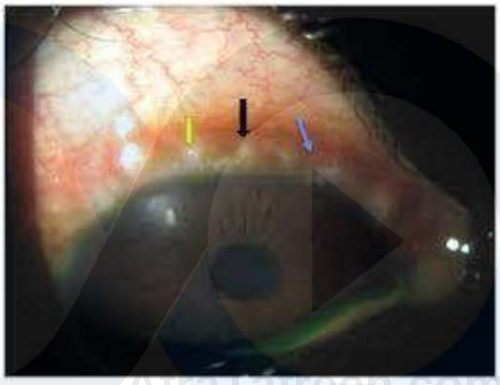
    Palpebral type of VKC
  - Limbal forms
    - Pseudogerontoxon
    - Horner tranta spots
    - Shield ulcer

Ropy discharge

Papillary form: -



Cobble Stone Papillae Fig: 1



VKC: Horner Tranta Spot Fig: 2

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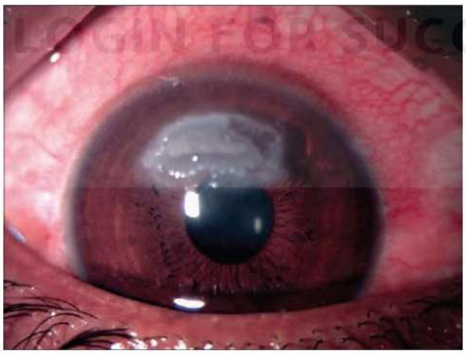
- These are eosinophils + cells over upper limbus



Shield ulcer Fig: 3

- Seen in upper ½ of cornea -

Pseudogerontoxon

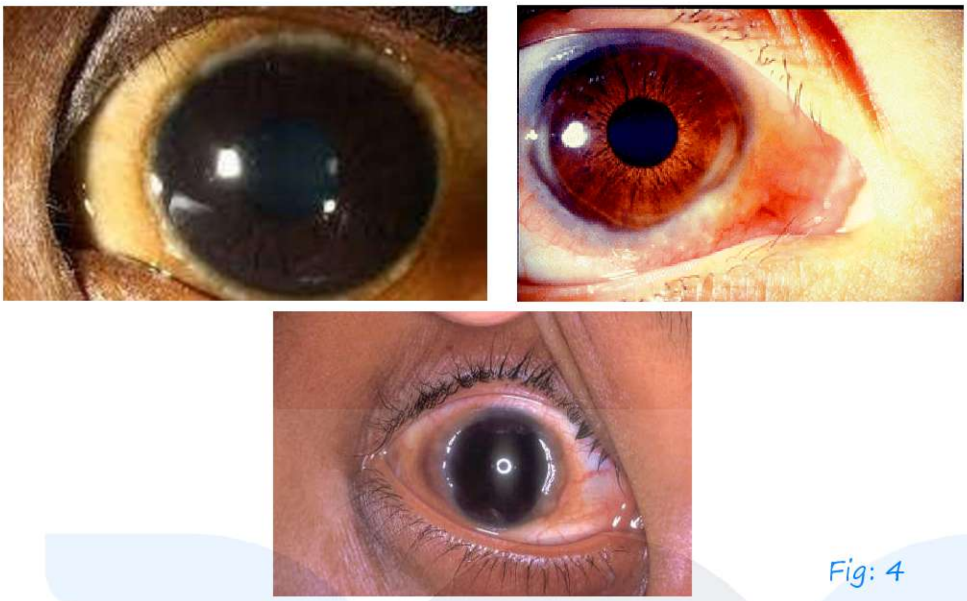


Fig: 4

- Exudation over limbus
  - Gerontoxon → Arcus senilis – corneal degeneration i.e., lipid accumulation around limbus seen in old age people
  - Pseudogerontoxon – looks like gerontoxon – seen in young children, VKC.

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Ropy discharge

- Right top

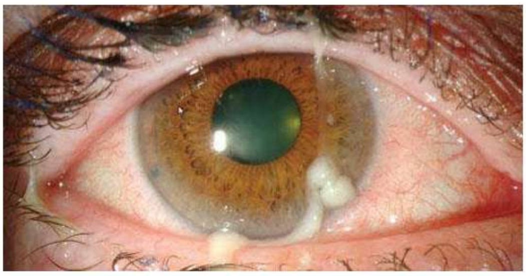
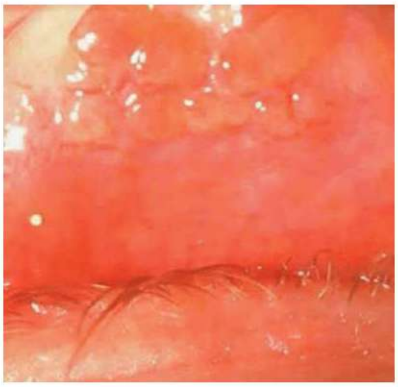


Fig: 5

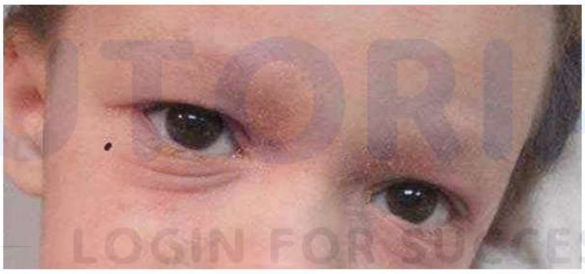
Maxwell lyon sign



Maxwell Lyon Sign Fig: 6

- Ropy discharge taking a form of membrane when exposed to heat – present in upper palpebral conjunctiva

Denny morgan line



Denny Morgan Line Fig: 7

- Extra fold of lower lid skin

Treatment 00:08:12

Symptomatic

- Anti-histaminic agents
- Mast cell stabilizers
  - Sodium cromoglycate

  - Ketotifen
  - Nedocromil
- Dual (1+2)

  Drug of choice
  - Olopatadine
  - Azelastine
  - Bepotastine
- Low dose steroid for initial few days
- Acetylcysteine – mucolytic agent
- Tacrolimus – ointment
- Cryotherapy for cobble stone papillae
- MNEMONIC: Boys have special sense of care, KEEP Relaxed, smiling dream for money
- Boys, horner tranta, summer, shield ulcer, Olopatadine, Cobble stone papillae, keratoconus, IgE, Exogenous, Pseudogerontoxon, Ropy discharge, steroid, Dennie morgan fold, Maxwell lyon sign.
- Keratoconus – due to frequent rubbing

Atopic keratoconjunctivitis (AKC) 00:12:27

- Variant of VKC
- Seen in young adults
- More severe compared to VKC
- Lot of skin problems
- Papillae are smaller in size

Giant papillary conjunctivitis 00:13:40

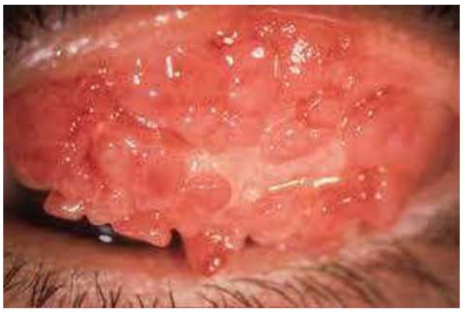


Fig: 8

- Papillae size - >1mm in size, over upper palpebral conjunctiva
- Type I, IV Hypersensitivity reaction
- Because of mechanical irritation of conjunctiva by
  - Soft contact lenses (most common)
  - Protruding suture
  - Scleral buckle in RD surgeries
  - Protruding bleb of trabeculectomy

Phlyctenular conjunctivitis 00:15:14

- Phlycten – Nodular elevation seen near limbus most commonly seen in the nasal part
- It is a type IV hypersensitivity reaction to endogenous allergen
- Most common allergen – protein of staph. Aureus in our country – Tuberculin hyper sensitivity
- Histopathologically – phlyctenules are sub epithelial inflammatory nodules containing histiocytes lymphocytes, plasma cells and neutrophils

- Sometimes this nodule undergoes necrosis and extends towards cornea and causes 3 types of ulcers

  1.Fasicular ulcer

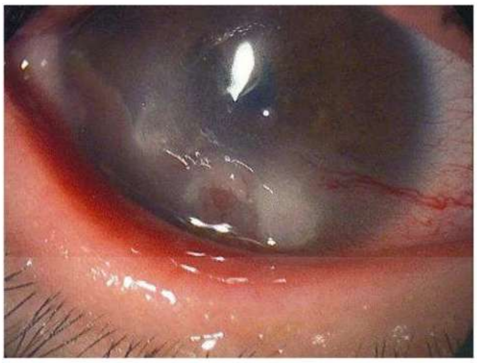


Fig: 8

– Leash of vessels coming within the corneal stroma

2.Sarcrofulous ulcer

3.Miliary ulcer

Treatment – Topical steroids –

If it is due to tuberculin – then start patient on ATT

DEGENERATION OF CONJUNCTIVA

Degenerations of conjunctiva 00:00:08

1.Concretions

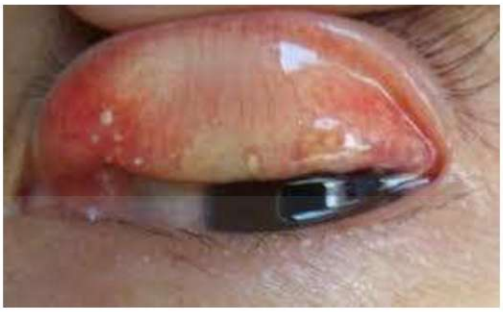


Fig: 1

- Mucous inspissated in glands of Henle causes lot of foreign body sensation

  Treatment – 26G needle – Take out the concretions

2.Pinguecula

Pingus – Fat

- Elastotic degenerative change in sub epithelial layer
- More prominent in nasal side
- Precursor of pterygium
- Lack of vascularization, corneal extension is differentiating feature of pinguecula from pterygium

3.Pterygium

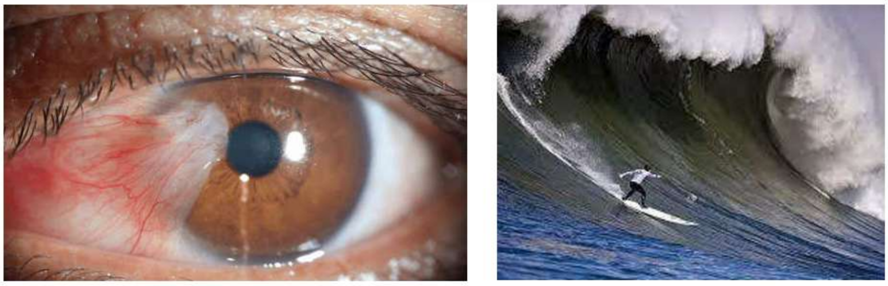


Fig: 2

- Definition – Wing shaped fold of conjunctiva which is encroaching on cornea
- P53 mutation → increase matrix metalloproteinase (MMP)
- More common in people exposed to sunlight (UV)
- Elastotic degeneration
- Most common – on nasal side

  When the pterygium is on both nasal, temporal side we call it as double pterygium

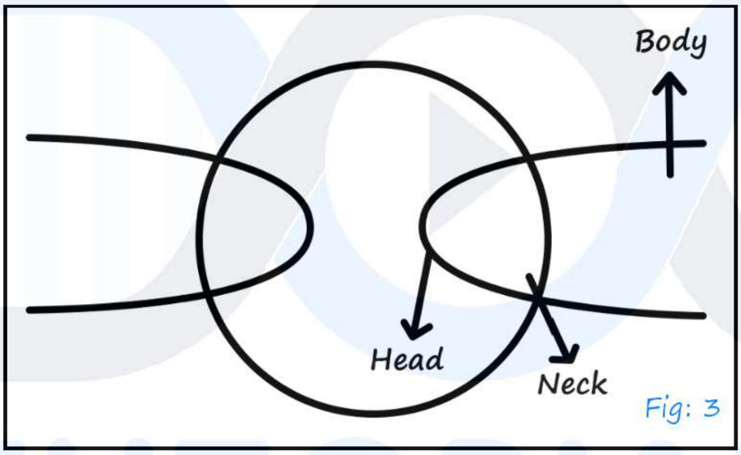


Fig: 3

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- Head

  i. Islet of vogt – White opacities (Epithelial cells)

  ii. Stocker's line

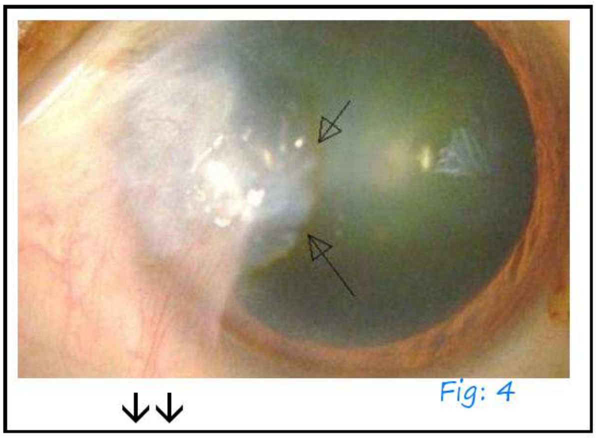

Fig: 4

↓↓

Accumulation of iron over head of pterygium

It is never seen in progressive pterygium

It is seen in stationary pterygium

iii. Fuchs flecks: At the leading edge of pterygium – collections of epithelial stems like cell

- Problems to patient
  - Cosmetic
  - Decrease in vision
    - Astigmatism – WTR
    - Encroachment on pupil
  - Diplopia

Treatment: - Surgery

1. Bare sclera technique
   60-70% Recurrence
2. Excision with mitomycin – c | 5 Fluorouracil
   These antifibrotic agents are applied after pterygium is excised.
3. Excision with autografting- best of all
   Graft can be taken from same eye or opposite eye
4. Perfect surgery
   - Pterygium extended resection followed by extended conjunctival transplant
   - Extensive tenon's removal
   - Large and thin graft (without tenon)
   - Avoid cautery / inflammation with glue

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Pseudo pterygium

- Mostly seen in post inflammatory eyes
- Can occur at any part of conjunctiva
- Probe can be passed under the neck of pseudo pterygium.

XEROPHTHALMIA

Spectrum of eye manifestations due to vit A deficiency

WHO staging of xerophthalmia

XN	Night blindness
X1A	Conjunctival xerosis
X1B	Bitot's spot
X2	Corneal xerosis
X3A	Corneal ulceration/ Keratomalacia (<1/3 corneal surface)
X3B	Corneal ulceration/ keratomalacia (≥ 1/3 corneal surface)
XS	Corneal scar
XF	Xerophthalmic fundus

- Night blindness is first symptom of xerophthalmia
- Xerosis is the first sign of xerophthalmia

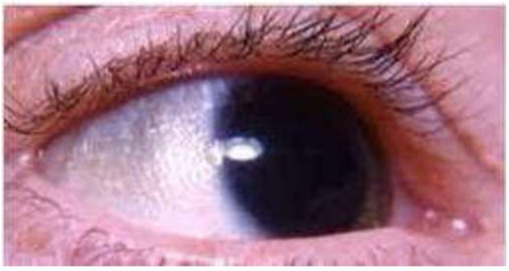

Fig: 1

- Conjunctival xerosis
- Histopathological

- Loss of goblet cells
- Squamous metaplasia
- Keratinization

After staining conjunctiva with Rose Bengal dye subtle dryness can be checked .

Bitot spots:

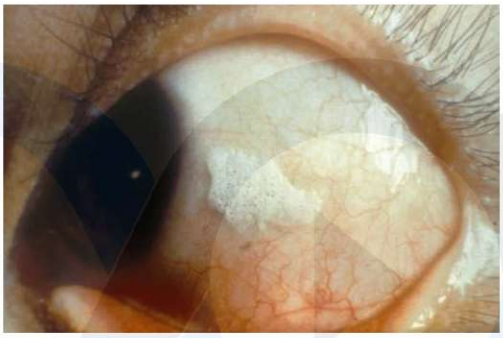

Fig: 3

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- Triangular foamy patch – whitish in color
  Apex towards – Lateral canthus
  Most common – on the temporal part of conjunctiva
- Foamy appearance is due to – fermentation by Corynebacterium xerosis
- If is a specific feature of xeropthalmia
- It appears late

Keratomalacia:

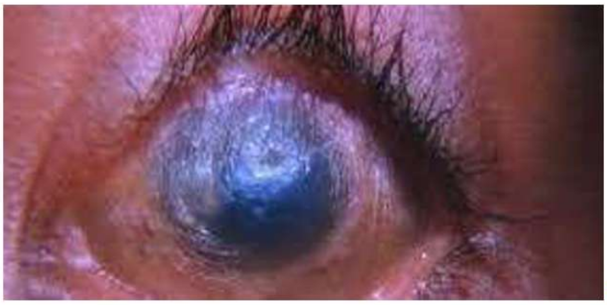


Fig: 3

- There will be corneal ulcer
- Necrosis
- Stromal melting can be seen

Treatment 00:04:05

Vitamin A Supplementation:

- If >1 year (or) 8 kg weight → 2 Lakh IU oral

Or

1 Lakh IU oral

If <1 year (or) <8 kg weight → 1 Lakh IU oral

Or

50,000 IU IM

These should by given for 3 doses and the schedule is 0, 1, 14.

DOC TUTORIALS
LOGIN FOR SUCCESS

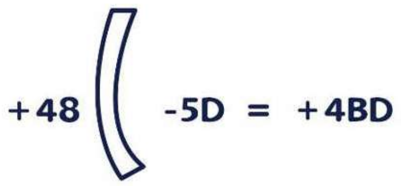
INTRODUCTION

- Anterior most covering of the eyeball
  To be more precise, anterior 1/6th of the outermost covering of the eyeball
- Transparent
- Avascular
  - Nourishment
    - Oxygen (From atruospheric air)
    - Solutes from aqueous humor and perilimbal capillaries (By diffusion)
- Refractive index
  - Aqueous, vitreous – 1.34
  - Cornea – 1.37
  - Lens – 1.39
  - Centre of lens – 1.42

  Total refractive power +60D

  +43 D (From cornea)

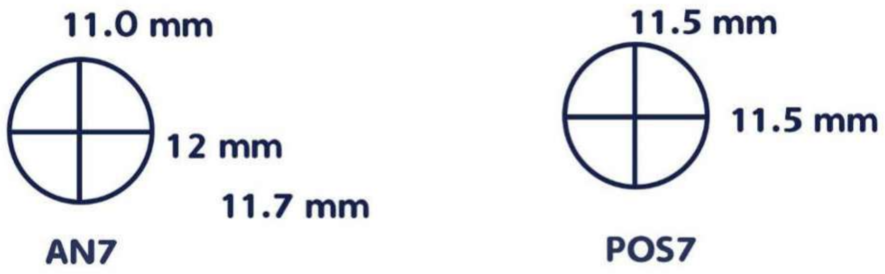
  +17D (From lens)



- Nerve supply
  - From trigeminal nerve (CN – V)

(Nasociliary Branch of ophthalmic division of trigeminal nerve)

- Dense nerve supply



Prolate in shape → steeper in the centre

Note:

a. Megalocornea
   - Corneal diameter > 13 mm (Horizontal)
   - Marfan syndrome, down syndrome

b. Microcornea
   - < 9mm at birth
     < 10 mm at any age after birth
   - Congenital rubella syndrome /congenital micro ophthalmia

Layers of cornea 00:06:55

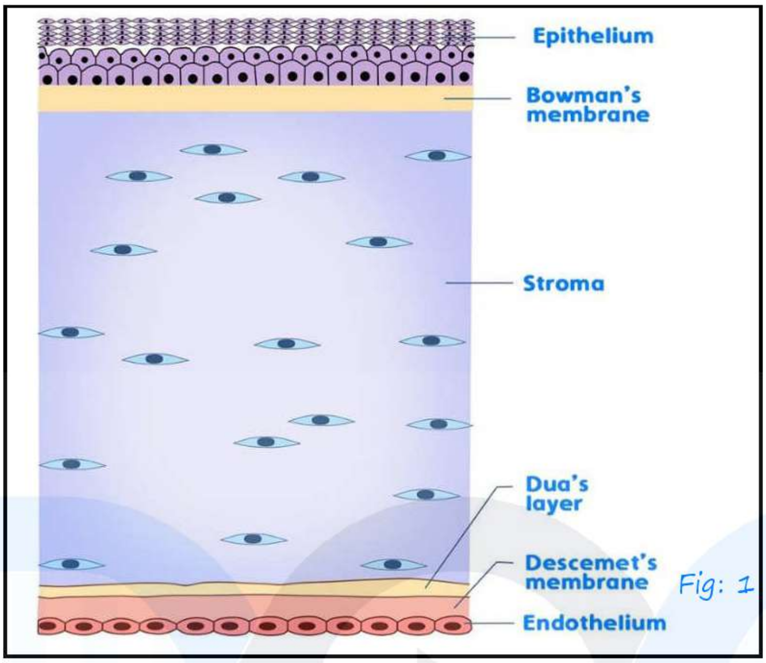

Epithelium
Bowman's membrane
Stroma
Dua's layer
Descemet's membrane
Endothelium

Fig: 1

a. Epithelium

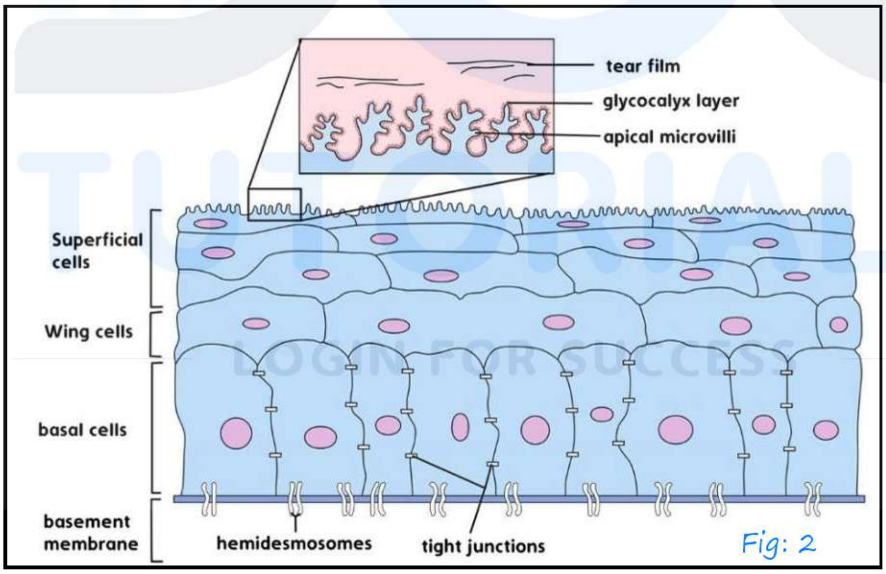

tear film
glycocalyx layer
apical microvilli
Superficial cells
Wing cells
basal cells
basement membrane
hemidesmosomes
tight junctions

Fig: 2

- 5-6 layered thick
- Basal layer
  - Columnar cells
  - Attached to basement membrane with hemidesmosomes

  - Clinical correlation: dystrophy of cornea
- Wing shaped cells

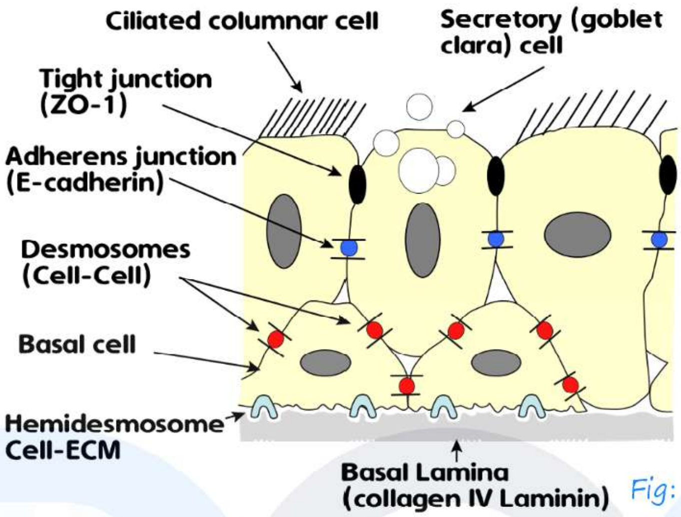

Ciliated columnar cell
Secretory (goblet clara) cell
Tight junction (ZO-1)
Adherens junction (E-cadherin)
Desmosomes (Cell-Cell)
Basal cell
Hemidesmosome Cell-ECM
Basal Lamina (collagen IV Laminin)

Fig: 3

  - Attached to basal layer by desmosomes
  - Tight junctions are seen between different types of cells

- Epithelium has regenerative properties (self-renewal) due to limbal epithelial stem cells in palisade of vogt (within the basement membrane)
- Assisted usually by patching the eye / BCL (Bandage Contact Lens)

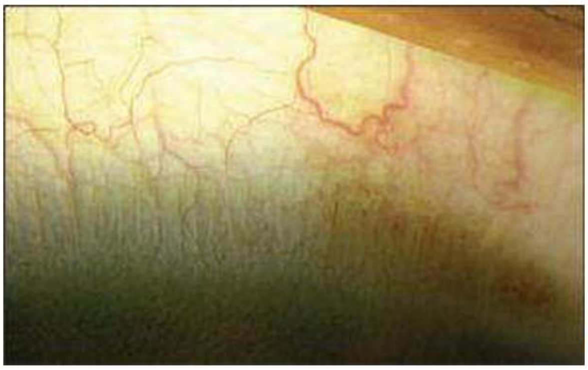


Fig: 4

- ABCG2
  - Specific limbal stem cell marker
  - Expressed in basal epithelial cells (not in others like supra Basal cells)
- p63 and vimentin → Present both in basal and suprabasal cells
- Epithelial turnover → 7-10 days for complete turnover of epithelium

Note

- Epithelium covered by tear film ~ 7 μ
- Superficial cells with apical microvilli – secrete glycocalyx which help tear film

b. Bowman's membrane

- False layer (Acellular – not a true membrane)
- Superficial condensed anterior stroma
- No regeneration

c. Stroma

- Maximum thickness (90% of corneal thickness)
- Secreted in 7th week of gestation
- Type 1,3,5 collagen → most common (Type-1)
  - Arranged parallel to each of them and to successive lamellae
  - Arranged in a lattice configuration
  - Distance between fibres is less than half of the wavelength of light, thus effectively neutralizing the scattering of light

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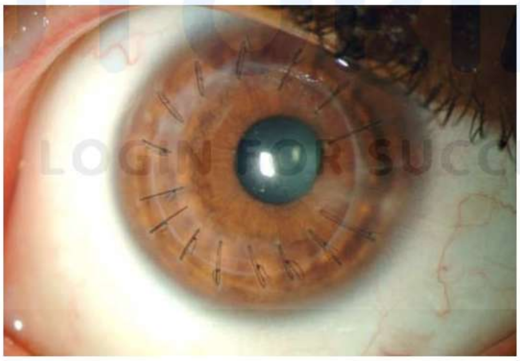
- Proteoglycans – regulate hydration of corneal strong
  - Keratin sulphate (most predominant)
  - Dermatan subphase
  - Chondritin sulphate
- Keratocytes
  - Cells of stroma
  - 5 % of volume of stroma
- Corneal crystallins – soluble proteins

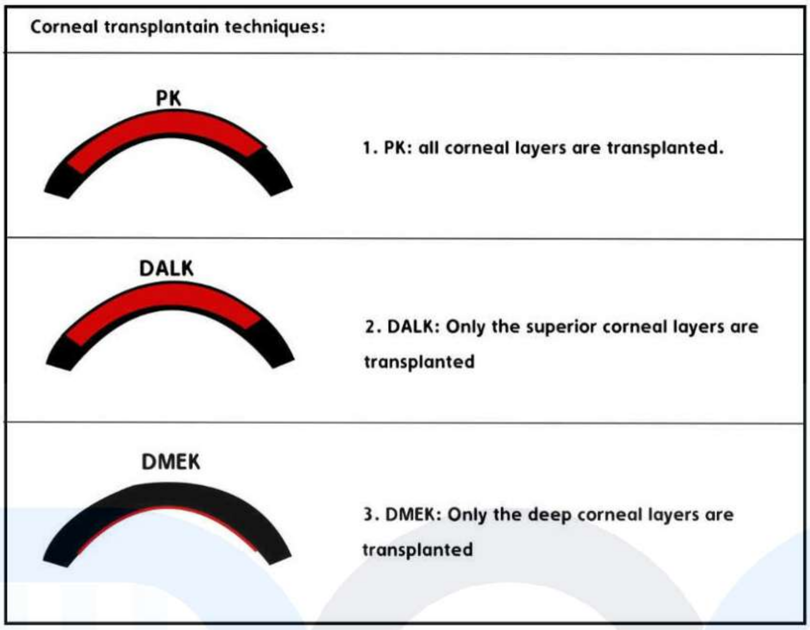
d. Dua's layer

- Newest layer
- Lamella
- 6-15 µ thick
- Most resistant layer of the cornea
- Splitting of cornea is done at this level



Penetrating keratoplasty Fig: 5

Lamellar keratoplasty


Corneal transplantain techniques:

1. PK: all corneal layers are transplanted.

2. DALK: Only the superior corneal layers are transplanted

3. DMEK: Only the deep corneal layers are transplanted

e. Descemet's membrane

- 3-10 μ thick
- Resistant layer
- Secreted continuously by the endothelium
- Starts by 8th week of gestation
- Type 4 and 8 collagen
- Very taut layer – No elastin

Trauma → thrown into folds towards the corneal stroma

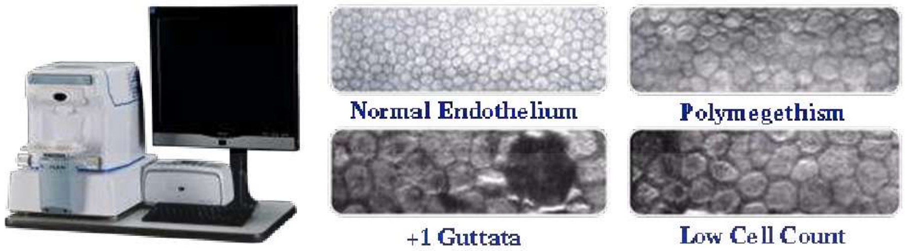
KF ring – Accumulation of copper

Schwalbe's line – end of Descemet's membrane on gonioscopy

Hassle Henle's body – wart like excrescence at the descemets membrane

f. Endothelium

- Innermost Layer
- Metabolically more active / numerous mitochondria
- 10 mm at birth → 5 mcm at adulthood
- Made up of single layer of hexagonal shaped cell


Normal Endothelium
Polymegethism
+1 Guttata
Low Cell Count

Specular microscopy Fig: 6

- Maintain corneal transparency
- $Na^+ K^+$ ATPase pump → Keeps cornea dehydrated
- 2500 cells / mm^2 as an adult
- Endothelial cell count is much higher in newborns 5000 – 6000 / mm2
- Loss of Endothelial Cells @ 0.3 % per year
- Counted using specular microscopy (Study and count number of endothelial cells)

If <500/mm^2 → decompensated cornea → plaques

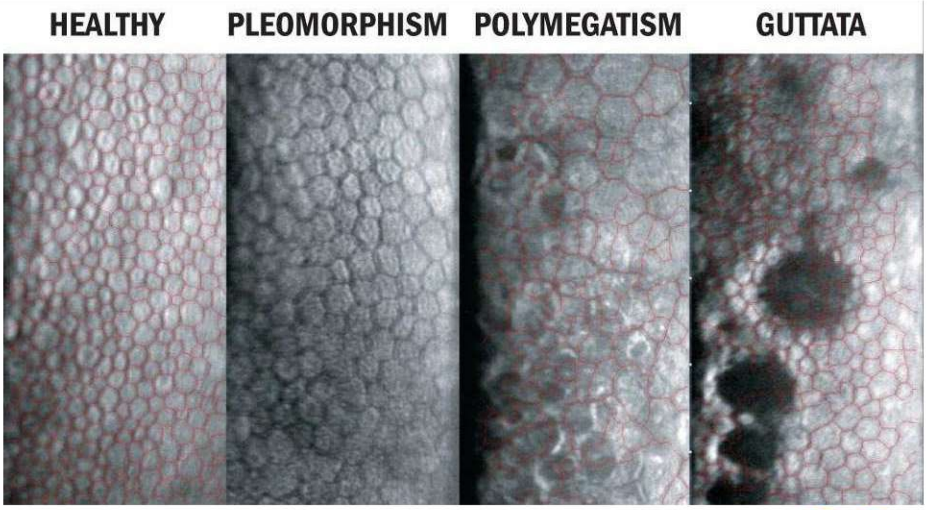


Fig: 7

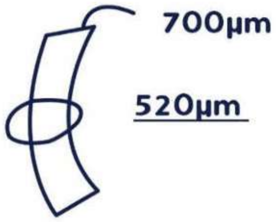
a. Pleomorphism – high variation in cell shape

b. Polymegation – high variation in size

c. Guttata – loss of endothelial cells

Reasons for corneal transparency 00:26:20

- Tear film
- Uniform and regular arrangement of corneal epithelium with good glycocalyx
- Peculiar arrangement of corneal stromal lamellae and the distance between them
- Corneal avascularity
- Physiological factors: relative state of corneal dehydration
- Normal LOP
- Endothelial pump (Na^+ K^+ ATPase pump)



→ CCT (Central corneal thickness)

Ultrasonic pachymetry

- Most common method, gold standard
- Probe → Piezoelectric crystal that emits an ultrasonic beam of 20MHz
- Transducer → It sends ultrasound rays through probe to cornea and receives echoes from cornea
- Faster, simpler and easy to use
- Consistent and repeatable
- Dry (no coupling medium required)
- Contact method

Manual optic pachymetry

Fig: 8

Keratometry 00:29:06

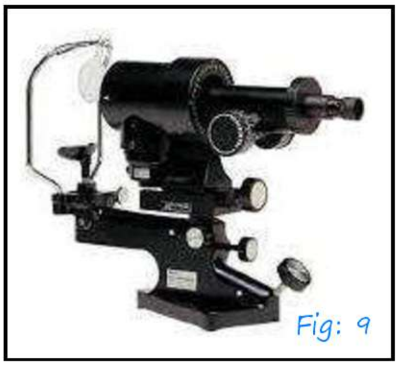


Fig: 9

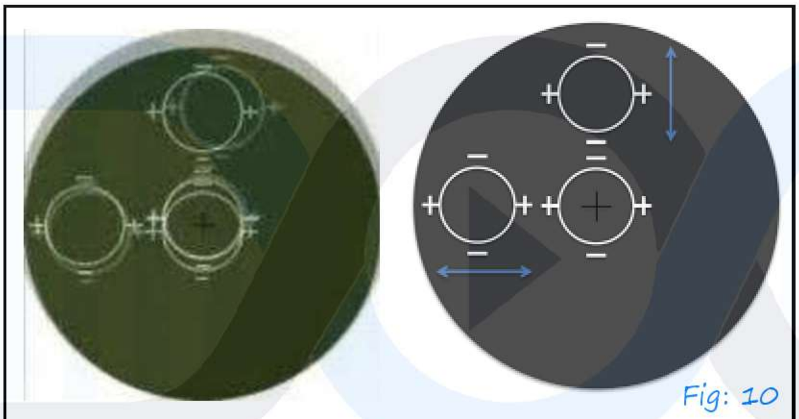


Fig: 10

Bausch and lomb keratometer

- Based on purkinje image
- Anterior surface of cornea – CONVEX MIRROR
- From Image Size formed by anterior surface of cornea (1st Purkinje image) – radius of curvature of cornea can be calculated
- Greater the curvature of cornea, lesser is the image size

Note

- Horizontally oval mires → WTR Astigmatism
- Vertically oval mires → ATR astigmatism

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Corneal sensations 00:31:48

- CN V – Trigeminal nerve
- Blink reflex – orbicularis oculi contract and CN VII
- Cotton wisp test

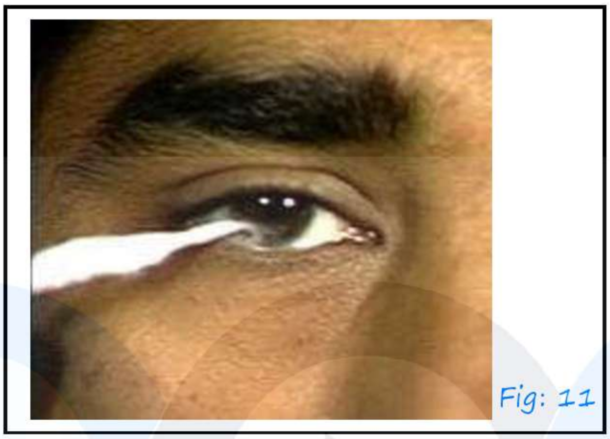

Fig: 11

- Touch the wisp on the corneal centre
- Ask for sensations apart from blinking
- Check in all quadrants (Superior, temporal, inferior, nasal)

Cochet - bonnet aesthesiometer

- Thin retractable nylon monofilament that extends upto 6 cm
- Variable pressure can be applied by adjusting the length
- 5 mm to 6 cm and as the length decreases, the pressure increases from 11 mm / gm to 200 mm/gm
- The shorter the length, lesser the sensation
- All the quadrants

Fig: 12

Decreased corneal sensations

- Herpes
- Diabetes Mellitus
- Ulcer
- Surgery
- Trauma
- Multiple sclerosis
- Cocaine
- Leprosy
- NSAIDs

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Causes of enlarged / prominent nerves

- MAN IRK GIRL

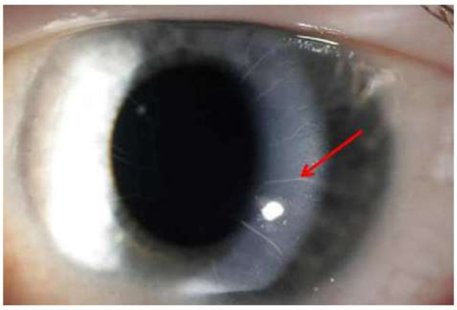

Fig: 13

a. MEN – I

b. Acanthamoeba
c. NeuroFibromatosis
d. Idiopathic
e. Riley day syndrome
f. Keratoeonus
g. Glaucoma (Congenital)
h. icthyosis
i. Refsum disease
j. Leprosy

Dyes 00:37:00

a. Fluorescein

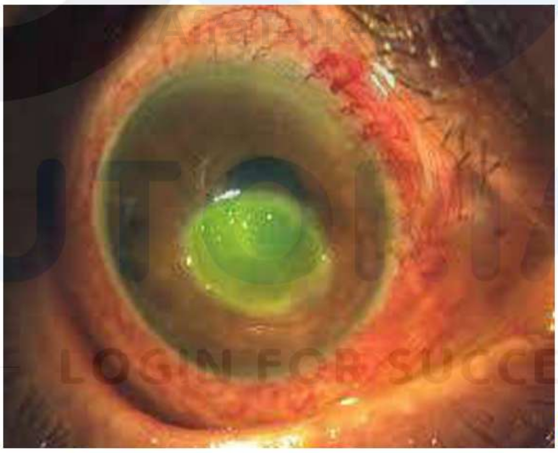

Fig: 14

- most commonly used
- Orange colored dye
- Stains epithelial defects green when viewed through cobalt blue filter

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b. Rose Bengal

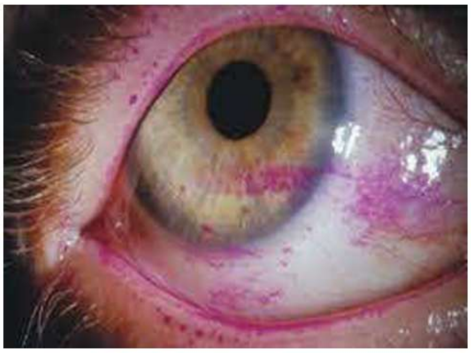


Fig: 15

- Pink coloured dye
- Dead devitalized cells
- Edge/margins of the ulcer stained

c. Lissamine green

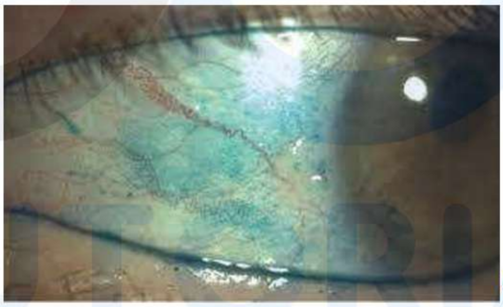


Fig: 16

- Stains corneal, conjunctiva
- Stains dead devitalized cells
- Does not stain healthy corneal surface

d. Alcian blue

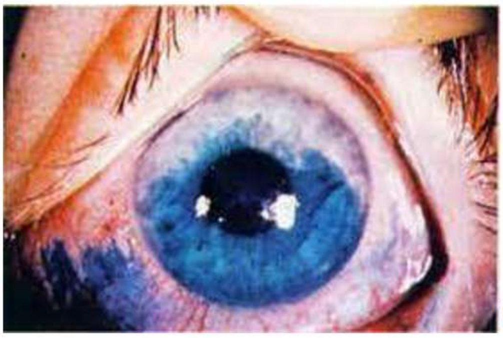
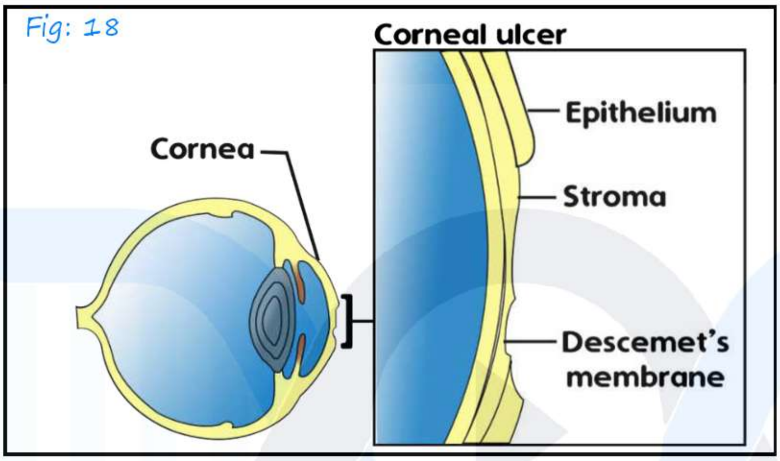


Fig: 17

- Muciln filament
- Irritant

Corneal ulcer 00:39:43

Fig: 18


Corneal ulcer
Cornea
Epithelium
Stroma
Descemet's membrane

- Epithelial defect with underlying necrosis and infiltration of stroma
- Prerequisite

Organisms penetrating intact epithelium

Mnemonic – contact lens safe nahi hai

- Corynebacterium
- Listeria
- Shigella
- Neisseria
- Haemophilus

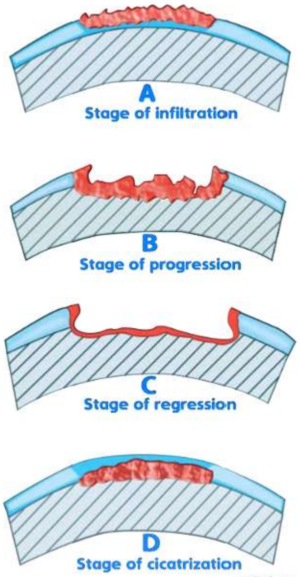

A
Stage of infiltration
B
Stage of progression
C
Stage of regression
D
Stage of cicatrization

Fig: 19

A. Stage of infiltration

- Epithelial defect
- Sausage shaped ulcer
- Overhanging ulcer marginn

B.Stage of progression

- Polymorphonuclear cell response

C.Stage of regression

- Appears as if ulcer size increase
- Necrotic material gets sloughed off

D. Stage of cicatrization

- aka stage of healing
- Reepithelization
- Vascularization ⊕ ⊕
- Decrease surrounding edema
- Opacity ⊕
- Pannus formation

Active corneal ulcer

- Typical cause of an acute red eye

Symptoms

- Pain (exposed nerve endings)
- Redness (Perilimbal / circumcorneal redness)
- Photophobia (not comfortable in ambient room light) → Toxic iridocyclitis
- Watering (Reflex – exposed nerve endings)
- Discharge (Superadded infection)
- Decrease vision

Most common cause of bacterial Ulcer

- Rest of the world: staph. Aureus
- India: Pneumococcus

Bacterial corneal ulcer

- Staphylococcus aureus
  - Oval
  - Sharp margins
  - Usually near limbus / paracentral
  - Skin infections / dacryocystitis
- Pneumococcus
  - Serpentine ulcer / hypopyon corneal ulcer (like a snake)
  - Has an advancing and trailing edge
  - Uveitis is common
  - Perforation can also be seen

Note:

- Other causative etiology + hypopyon → corneal ulcer with hypopyon
- Pneumococcus → Hypopyon corneal ulcer

- Moraxella
  - DM
  - Drug abuse
  - Immunocompromised
  - Paralimbal / limbal ulcer
- Pseudomonas
  - Biofilm ⊕
  - most commonly seen in contact lens users
  - Green coloured discharge
  - Early perforation
  - Ring abscess

Atypical mycobacteria

- Post operative / post LASIK ulcers

Klebsiella / E. coli

- Post traumatic / compromised cornea

Nocardia

- Resembles fungal ulcer
- Cracked windshield appearance
- Satellite lesions

CORNEAL ULCERS

Complications in corneal ulcer

- Deeping ulcer sticking Descemet's membrane → protrusion/folds/prolapse → descemetocele
- Cough/stain → rupture through & through perforation → increase IOP
- Perforation :

  1. small

  iris migrates towards the defect and plugs it

  → anterior synechiae

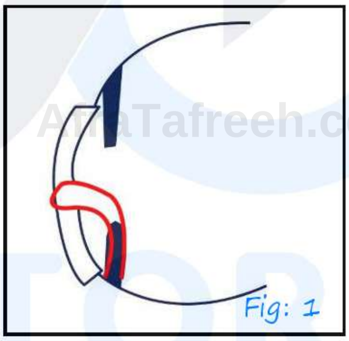


Fig: 1

- Exudation /inflammatory processes

  - Pseudo cornea

  2.Large perforation

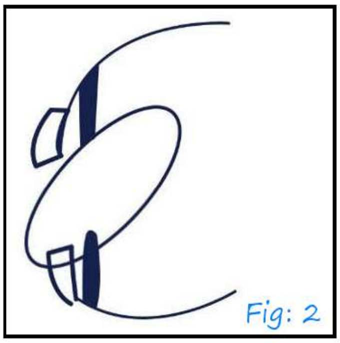


Fig: 2

- Lens gets subluxated
- Endangers globe integrity

Treatment

- Tectonic penetrating keratoplasty
- Anterior staphyloma formation

Treatment of bacterial corneal ulcers

- Topical antibiotics
  - Fortified → higher concentration
  - As combination of gram positive /gram negative
  - 50 mg/ ml → 5 % { cephazolin tobramycin, ceftazidine vancomycin
  - 4th gen FQ → moxifloxacin
  - Available in lyophilizate form /powdered 500 mg /2ml
  - Dilated with 8 ml of eyedrops like R. L
- Cycloplegic agents (atropine like drugs)
- State of sustained contraction of ciliary muscles gets relieved
- Atropine ointment (longest and strongest acting)
- Alternates – homatropine
  - Cyclopentolate
- if IOP is more
  - topical drugs will not be able to act in cases of IOP
- Perforation
  - Small – BCL (bandage contact lens)

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- Large – PK (tectonic keratoplasty)

Fungal ulcer 00:09:00

- History of trauma by vegetative /organic material/ tail of animal
- History of steroid usage

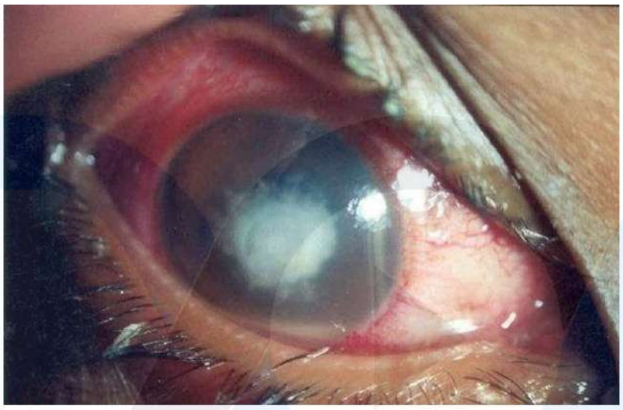
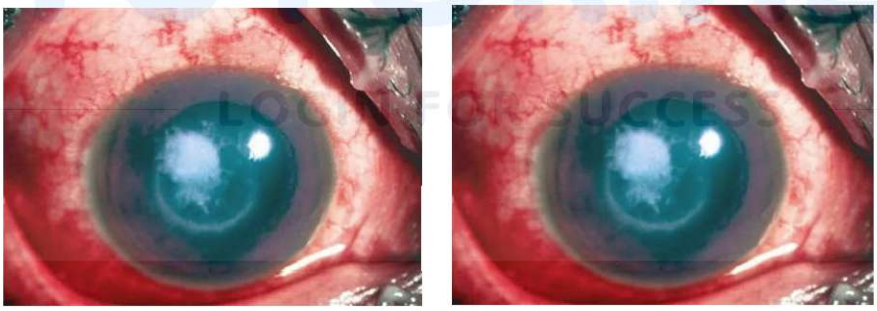


Fig: 3

- Most common → aspergillus (m/c)

  → Candida /yeast most common



Ring of weissley

Fig: 4

Symptoms less compared to signs

- Signs

  a. Feathery margins classical for fungal ulcer

  b. Dry looking ulcer

c. Satellite lesions

d. Fungal hyphae will go around and cause multiple small ulcers in vicinity of the main ulcer

e. Ring shaped ulcers
(Immunological ring of weissley) → can also be seen in pseudomonas, acanthamoeba

f. Perforation is very rare

g. Hypopyon → fixed and unsterile (presence of fungal hyphae)

Diagnostic procedure scraping

- Specimen from the base and the edge of the ulcers
- Sample for screening (KOH mount)

In vivo confocal microscopy

- Resolution of 1-2 μ
- Non invasive
- Fungal hyphae
- Cysts and trophozoites of acanthamoeba → seen on microscopy

Treatment of choice

- 5% natamycin topical
- Cycloplegic are also to be given(to relieve pain)
- If ulcer is deep → voriconazole (topical 1-3%) intrastromal

→ Posaconazole (oral 200 mg) twice a day

- C3R → antifungal activity is present
  → it halts deeper progression

Viral ulcers 00:19:10

- Herpes simplex virus
  - Decreases the corneal sensation
  - Recurrent corneal ulcers
  - 1° → non immune /children → skin is affected
  - 2° reactivated → belpharoconjuctivitis
    → keratoconjunctivitis

HSV- epithelial involvement

- Superficial punctate keratitis (SPK)
  - Can cause photophobia /pain in patients
  - Decrease vision
  - Self-limiting /tear drops

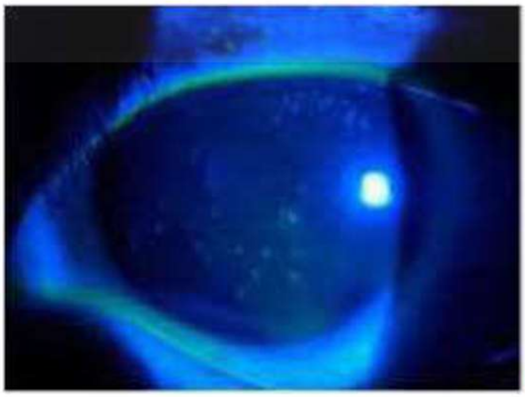


Fig: 5

- Dendritic ulcer
  - Superficial keratitis (epithelial)

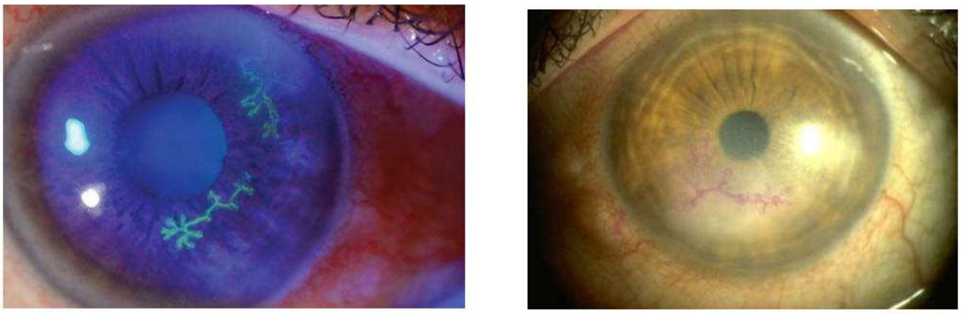


Fig: 6

  - Dichomatous branching (visible on fluorescein dye)
  - True dendrite – knobs at the end always
  - Note →margin of ulcer is stained by Rose Bengal dye
  - Steroid is an absolute contraindication (can cause geographical ulcers)
  - Treatment of choice : 3 % acyclovir ointment
- Geographical ulcer
  - All the dendrites get joined together and form a huge area of ulcer on the cornea

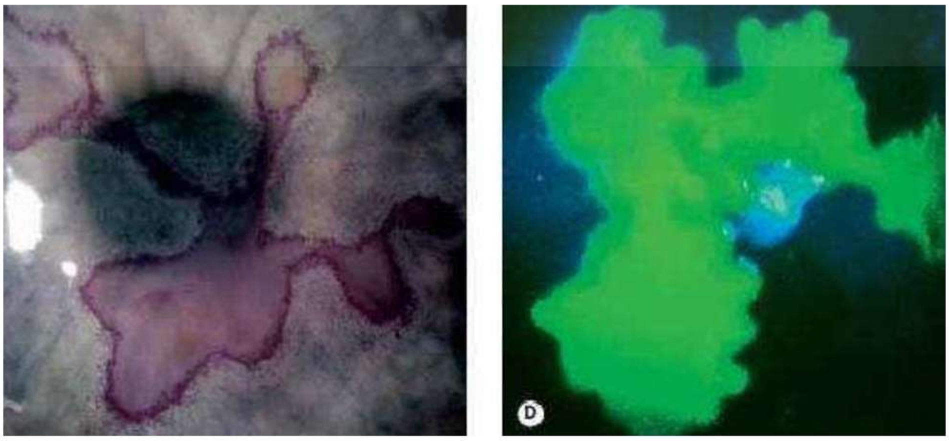


Fig: 7

HSV → stromal involvement

- Viral antigen may be present but replication may not be there
- Underlying necrosis → tissue destruction

Treatment

- Topical steroids under the cover of antigen

HSV → Disciform keratins /endothelitis

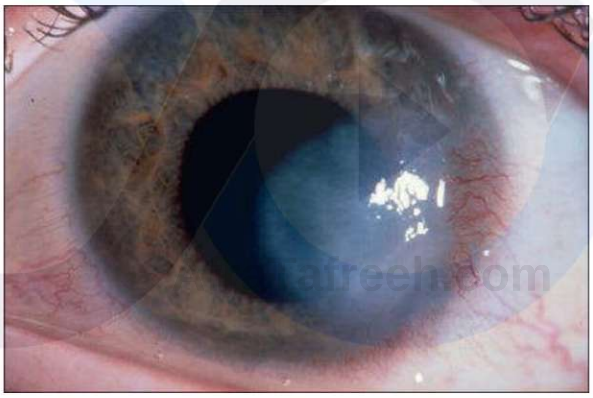


Fig: 8

- Past history of dendritic ulcer is not always there
- May be a hypersensitivity reaction to a viral antigen or HSV infection of keratocyte or endothelium
- Lot of DM folds stromal edema/ulcers

Treatment

- Topical steroid under the covers of antiviral agents

HSV→ Metaherpetic ulcer

- No active virus but a problem with the epithelial healing
- Heaped up edges of the epithelium seen
- Treated using adequate quantities of tear supplements

Herpes zoster opathalmicus 00:26:30

- Virus in the sensory ganglion (reactivated)
- Comes along trigeminal nerve – ophthalmic division /nasociliary nerve

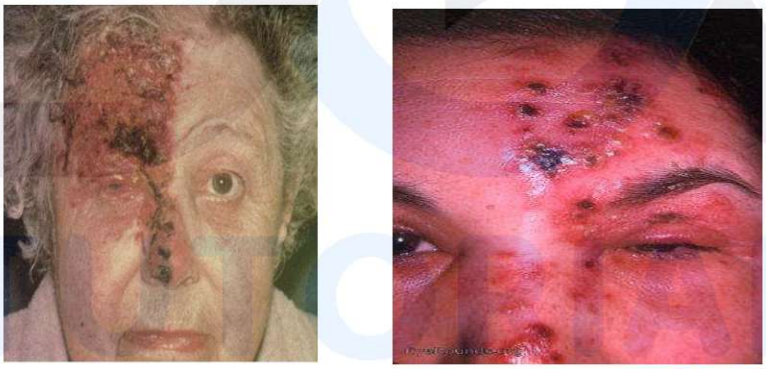


Fig: 9

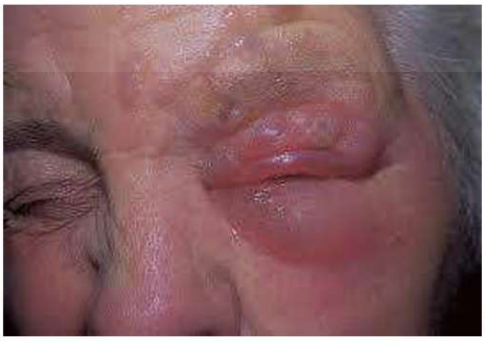


Fig: 10

- Hutchison rule
- Predicts the ocular involvement

- If the tip of the nose has vesicles, high chance of involvement of eye /cornea
- Common nerve supply (nasocilairy nerve)
- Most common nerve ininvolv-Frontal division of trigeminal nerve
- Treatment -oral acyclovir 800 mg 5 times a day for 14 days

Pseudo dendritic keratitis

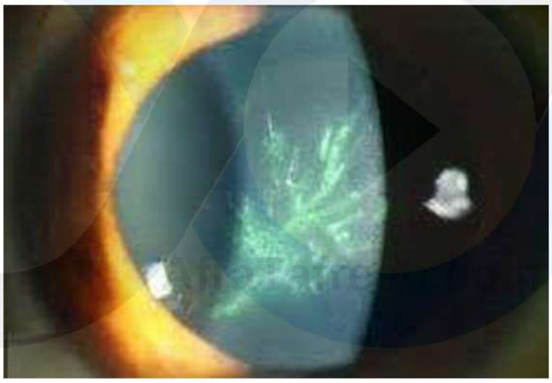


Fig: 11

- Absence of terminal knobs

Nummular keratitis

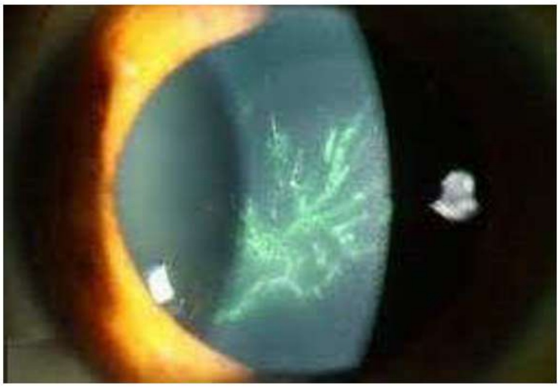


Fig: 12

- Small coin shaped opacities
- Subepithelial infiltrates
- Photophobia
- Decrease in vision
- Note – conjunctivitis/ scleritis (rare, optic nerve involvement /retinochoroiditis/ uveitis

Acanthamoeba keratitis 00:31:36

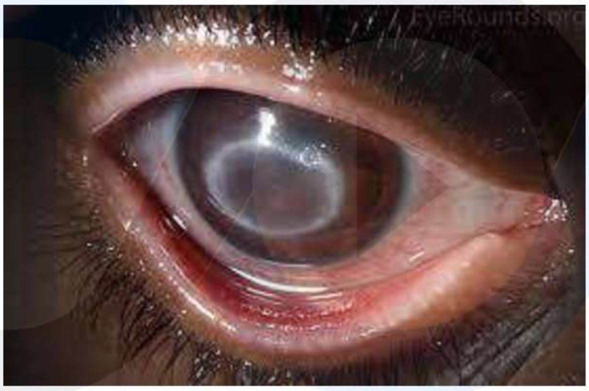


Fig: 13

- Free lying protozoa in the soil
- History of bathing in dirty water, pools unsterile solution for contact lens applications
- More specific in contact lens users
- Most painful – cause radial keratoneuritis (inflammation of corneal nerve endings)
- Mimics others ulcers (very difficult to diagrams)
- Note: Pseudomonal most common in contact lens users

Investigation

- confocal microscopy
- Non nutrient agar enriched with E. Coli → growth of trophozoites
- DOC: -

  a. Biguanide – poly hexamethylene biguanide

  b. Chlorhexidine

  c. Propamidine

  d. Antifungal agents

Trophic ulcers 00:35:08

Neurotrophic ulcer	Neuroparalytic ulcer
• Problems in sensory nerve supply (Cranial nerve V palsy)	• Cranial nerve VII palsy

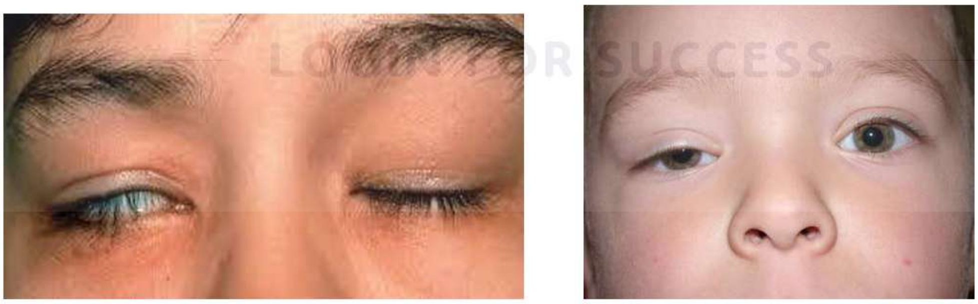


Fig: 14

Lagophthalmos

- Effect in nerve supply /transmission to orbicularis oculus

- Corneal exposure (more in lower half)
- Exposure keratopathy /neuroparalytic ulcers
- Treatment - patch eye/tear supplements /suture the eyelid in paramedian part (surgical tarsorrhaphy)

Interstitial ulcers (stromal ulcers)

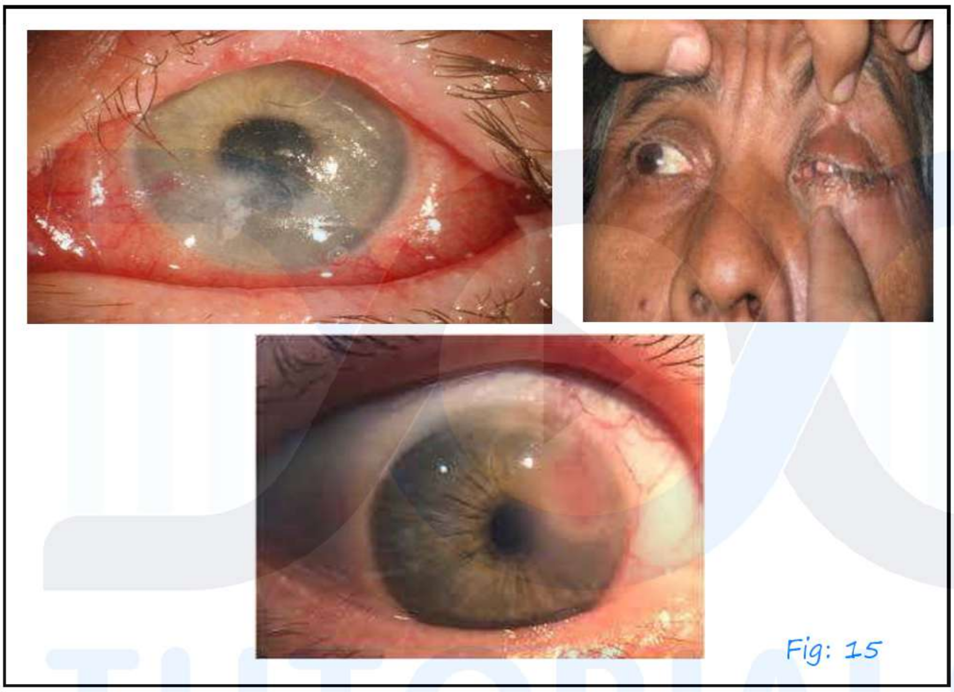


Fig: 15

- Immune mediated hypersensitivity reaction
- Epithelium and endothelium are normal
- Most common in congenital syphillis
- Tb, leprosy, Lyme's disease, sarcoidosis, cogan's syndrome- (interstitial keratitis + sensory neural deafness)
- Tongue shaped patch of pink color vascularized stroma -slamon patch)

Peripheral Ulcerative keratitis 00:41:02

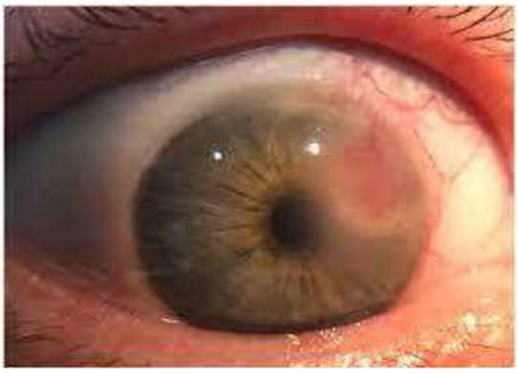


Fig: 15

- Peripheral part
- Ulceration
- Associated with autoimmune – presented with steroids usually (RA, PAN, SLE)
- Sclera can also be involved
- Limbus may be spared

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Mooren Ulcer

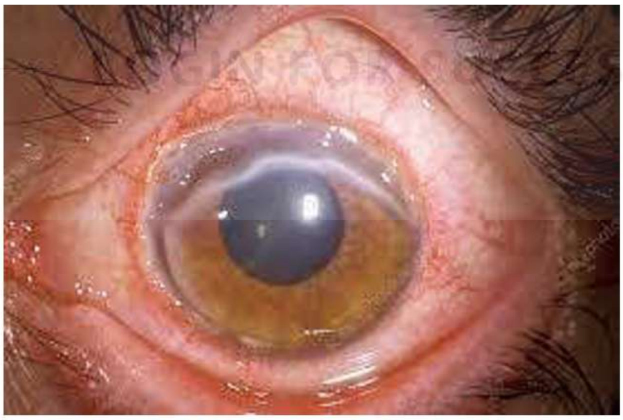
Fig: 16

- Idiopathic
- Undermined edges
- Progressive stromal melting

- autoimmune +/-
- Limbus affected
- Type 1 unilateral/mild /old
- Type 2 bilateral /aggressive /young
- Type 3 indolent

Terrine's marginal degermation

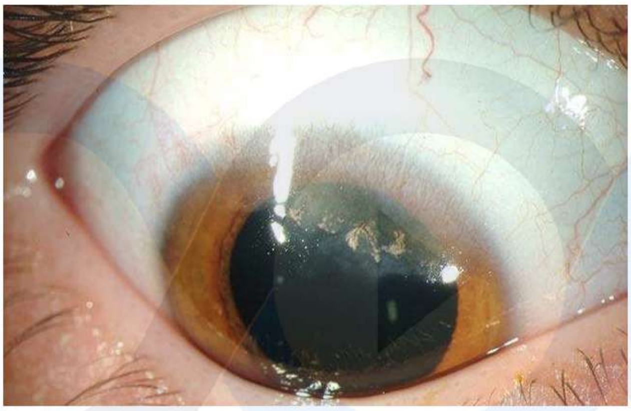

Fig: 17

- Not painful
- Slowly progressive non inflammation, peripheral cornel thinning and is associated with corneal neovascularization opacification and lipid deposition
- ATR, astigmatism
- Differentiate form moorens because there is no pain/inflammation the epithelium is intact and there is no overlying edges

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CORNEAL DYSTROPHIES AND DEGENERATIONS

Degeneration	Dystrophy
Late in age	Usually at birth
UL/BL	Mostly BL
Peripheral, Asymmetrical	Symmetrical
Associated with systemic disease, ageing	AD, no systemic association, family history may be positive
May not affect vision	Vision is impaired later severely

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Corneal dystrophy

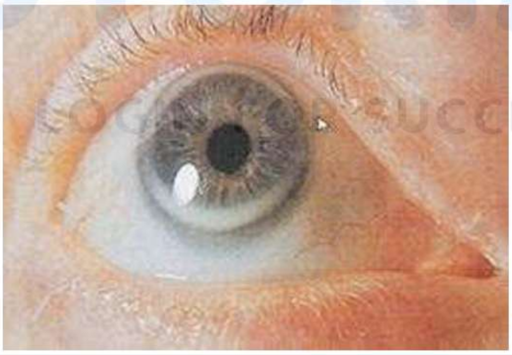

Fig: 1

- Early age of onset
- More common in central part of corneal
- Progressive loss of vision

Types of Corneal Dystrophies

1. Epithelial
2. Bowman's membrane
3. Stromal
4. Endothelial

Epithelial dystrophy 00:02:01

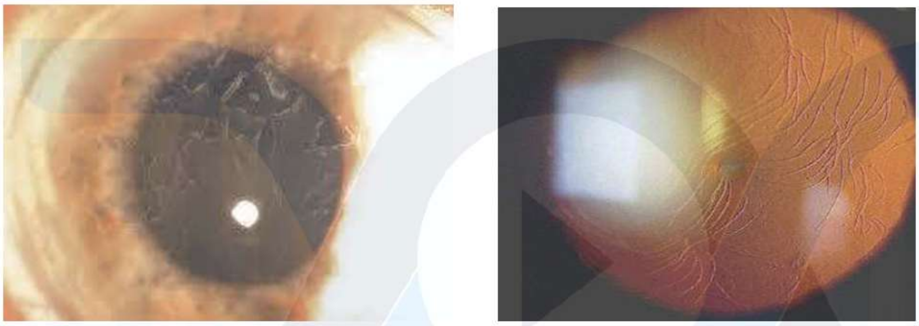

Fig: 2

EBMD (Cogan's Microcystic Dystrophy)

- Epithelial basement membrane dystrophy
- At end of 1st decade/start of 2nd decade
- Defects in the Hemidesmosomes (Recurrent spontaneous corneal erosions)
- Also called as Map Dot / Fingerprint dystrophy)
- Treatment: Scrape off the epithelium

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Meesman epithelial dystrophy 00:04:11

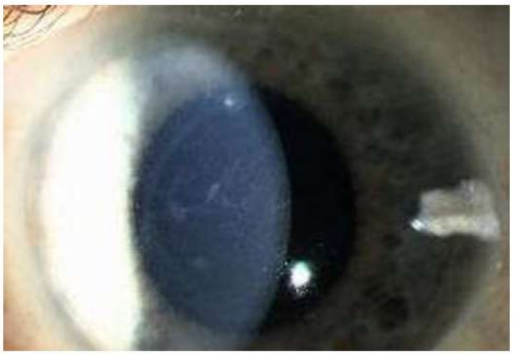

Fig: 3

- Occurs early in life
- Intraepithelial cyst formation

Bowman's dystrophy 00:04:40

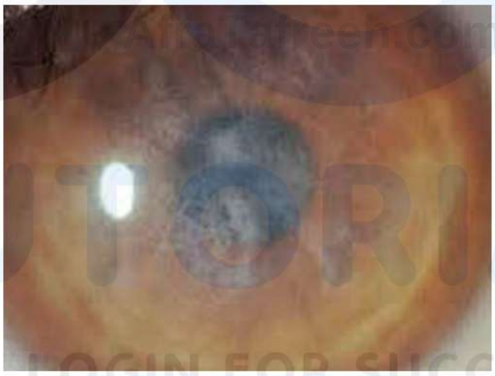

Fig: 4

- Reiss Buckler (Autosomal Dominant)
  - Grey white polygonal opacities at the level of bowman's membrane
  - Replacement of bowman's membrane with fibrous tissue
- Theil Behnke dystrophy (Autosomal Dominant)
  - Similar opacities but with a honey comb pattern

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Stromal Dystrophy 00:05:14

- Marylyn Munroe always gets her man in LA club (mnemonic device)
  a. Marylyn Munroe always
    - Macular: Mucopolysaccharides, Alcian blue (colloidal iron)
    - Least common AR; worst prognosis
  b. Gets her man
    - Granular: Hyaline (Masson's trichome)
  c. LA club
    - Lattice: amyloid (Congo red) → most common; AD; best prognosis

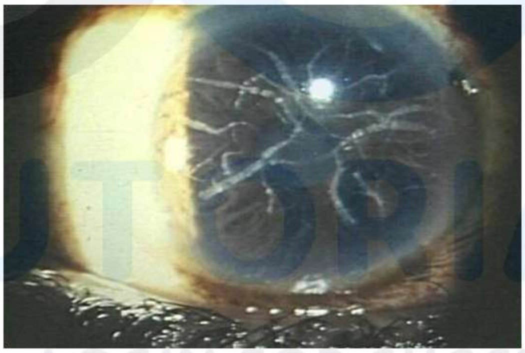

Fig: 5

- Criss cross deposition of amyloid the level of stroma

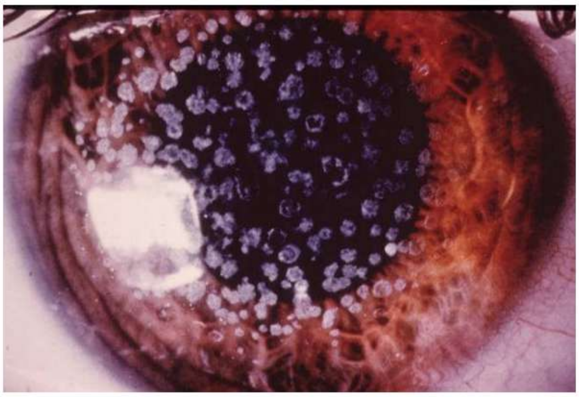

Fig: 6

- Popcorn like depositions of hyaline with clear areas in between

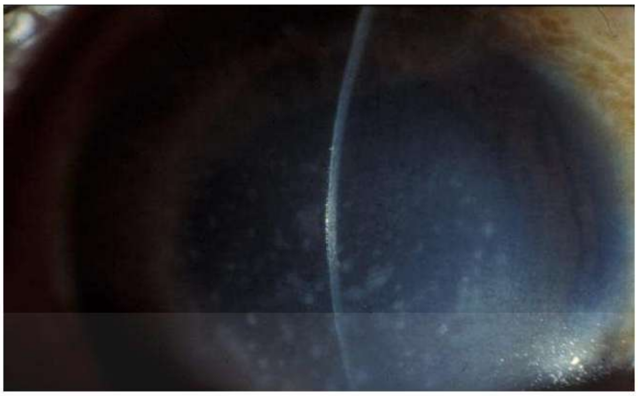

Fig: 7

- Edema; white out spread from one limbal border rot another spared
- No clear intervening areas

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Endothelial Dystrophy 00:08:10

1. Fuch's endothelial dystrophy
   a. Seen in elderly females
   b. Pain in the eye is worse when they wake up in the morning
   c. Loss of endothelial cells – Guttae
   d. Increases with trauma/open eye surgery
   e. High association with primary open angle glaucoma
   f. Sporadic is the most common etiology
   g. Autosomal dominant forms can also be seen
   h. Bronze metal appearance on the inner sickle of corneal slit

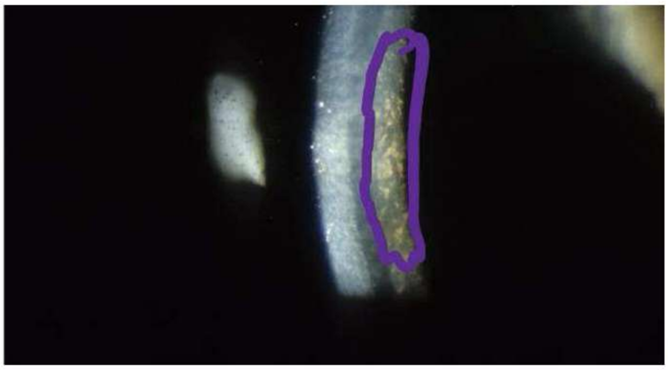


Fig: 8

i. **Progression**

  i. **Guttae** – Involve stroma, come into epithelium and gives rise to oedema

  ii. **Central oedema** – Hazy vision

  iii. Rupture

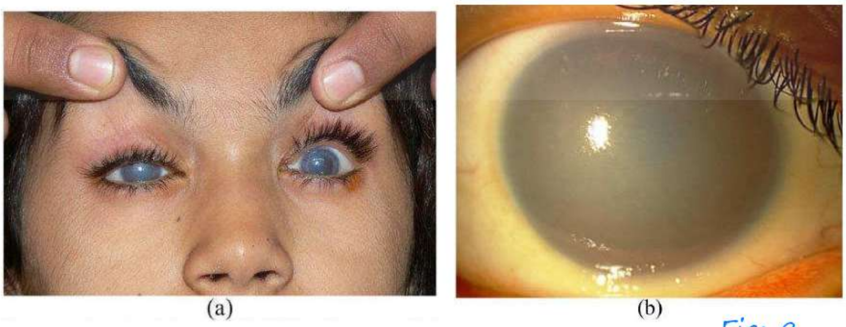
j. **Management**

  i. Control IOP

  ii. Replace corneal

  iii. DMEK/lamella keratoplasty

2. Congenital Hereditary Endothelial Dystrophy (CHED) 00:10:27



(a) (b)

Fig: 9

a. A differential for cloudy corneal at birth

b. Generalized absence of endothelium

c. Grounds glass appearance

Note:

STUMPED (mnemonic) → cloudy corneal at birth

- Sclera corneal
- Birth trauma
- Corneal ulcer
- Mucopolysaccharides
- Peter's anomaly
- Endothelial cyst
- Corneal dermoid

3. Posterior polymorphous dystrophy (PPD)

00:12:00

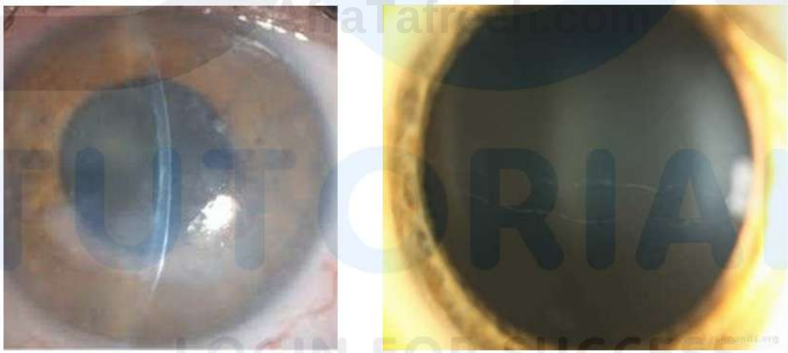


Fig: 10

a. Endothelium becomes like epithelium/multilayer

b. Corneal oedema and thickening'

c. Glaucoma can also occur

d. Snail track lesion in a band form

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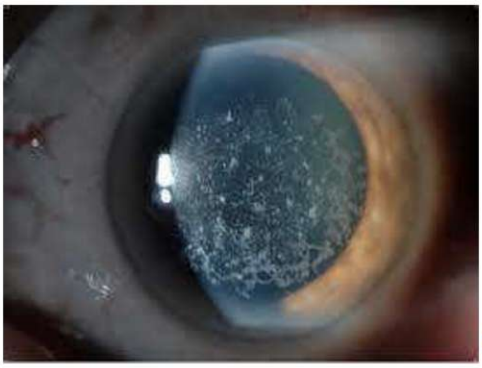

Fig: 11

1.Arcus senilis

- With Age
- Doesn't affect vision
- Associated with hyperlipoproteinemia type 2
- Accumulation of lipid in an arc/circle
- Vogt's interval → gap between Arcus and limbus
- Unilateral arcus is seen in ocular hypotony /aroid artery disease

Arcus Juveniles

- In young age

2.Climatic /droplet keratopathy/actinic keratopathy /Bietti

a. Golden granules seen as degermation in the corneal stroma

b. Seen in mostly in men who are working in sun light

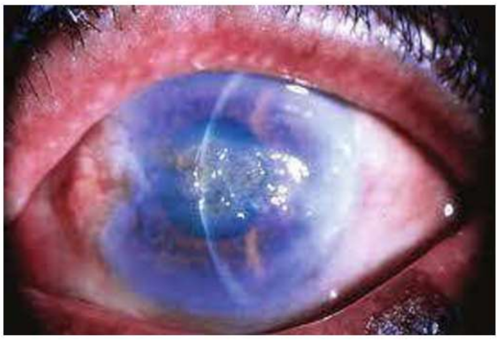

Fig: 12

4. Saltzman nodule degermation 00:15:09

a. Appearance of small nodules

b. Discrete grey nodular superficial stromal opacites

c. Fragmented /absent BM

d. Meibomian gland dysfunction is present

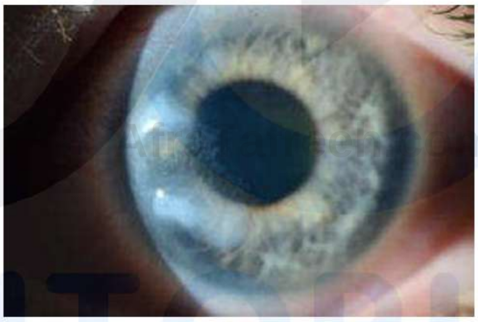

Fig: 13

5. Lipid degeneration 00:15:44

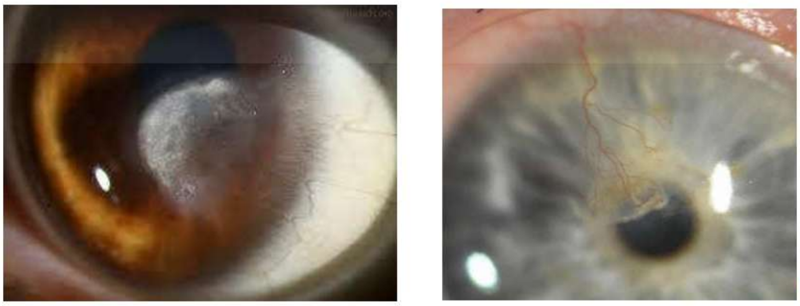

Fig: 14

A. Primary
  a. Less common
  b. Not related to elevated serum lipid levels
  c. Vascularization definitely absent
  d. Whitish patch near the periphery of the corneal
  e. There's hardly an interference with vision
  f. Superficial opacity

B. Secondary
  a. More common
  b. Associated with corneal neovascularization
  c. Associated with previous ocular injury or pathology

6. Band shaped keratopathy 00:16:34

a. Accumulation of calcium as a band in superficial stroma in the interpalpebral region

b. Treatment → EDTA Chelation

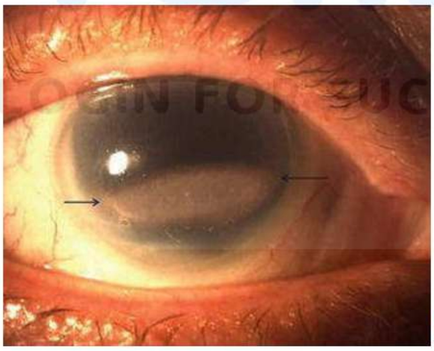

Fig: 15

CUT PGS (mnemonic)

Hyperkalemia	Phthisis bulbi
Ulcer/uveitis	Long standing glaucoma
Trauma to the eye	Silicone oil in AC (more common in adults)

7. Corneal verticillata 00:18:25

a. Broomstick like accumulation in cornea (bowman membrane/ stoma) → migration of epithelial cells

b. Mostly associated with chronic usage of drugs

c. Also called as vortex keratopathy /whorl spared hematophagy

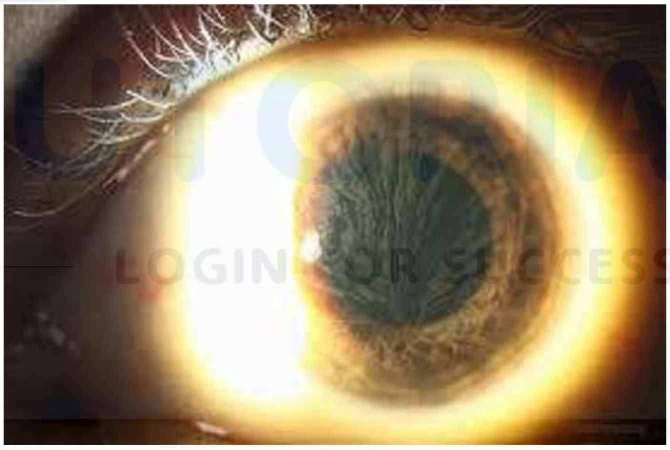

Fig: 16

Fitness (ARD (mnemonic)

1. Fabry's disease
2. Indomethacin
3. Tamoxifen
4. Chloroquine, Chlorpromazine (phenothiazine)

5.Amiodarone (most common)

6.Rho kinase inhibitors

7.Diclofenac

It is reversible on stoppage of drug use

Ectatic conditions of Cornea 00:20:50

a. Keratoconus

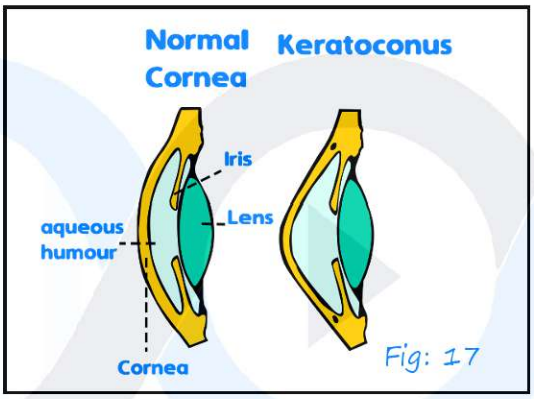
Normal Cornea
Keratoconus
Iris
Lens
aqueous humour
Cornea

Fig: 17

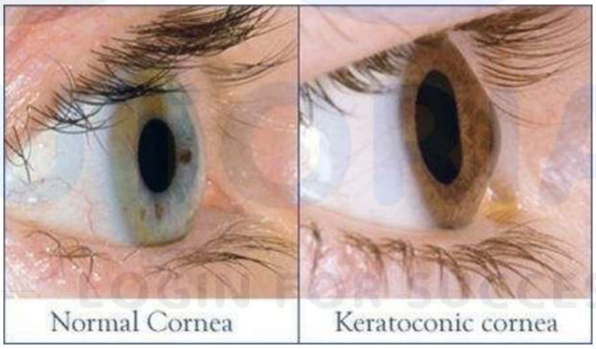
Normal Cornea
Keratoconic cornea

Fig: 18

- Bilateral asymmetric/symmetric
- Idiopathic progressive ectasia
- More commonly in young females
- Progression gets arrested in the age of 30s
- Unable cross linkage in the corneal stroma

- Progressive or frequent change in glasses' power (With-the-rule → irregular astigmatism) (due to scarring)

Signs

Mnemonic - Keratoconus OF MR VANSH

A. Oil droplet reflex

   a. Globular appearance though ophthalmoscopy

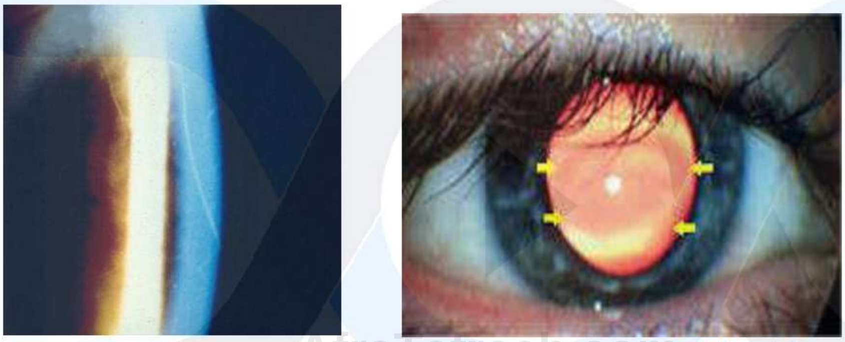

Fig: 19

B. Fleischer ring

   a. Iron ring on the base of the cone

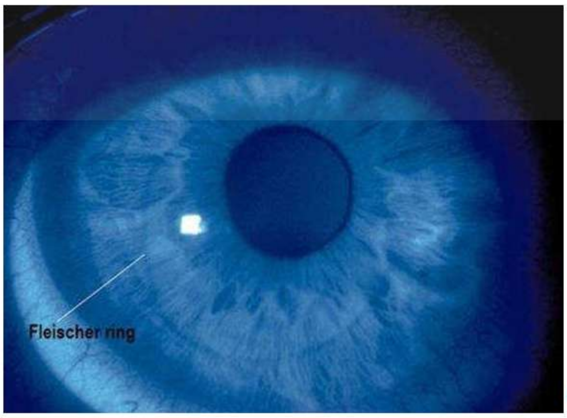

Fig: 20

C.Munson sign

a.Notching on lower lid on looking down

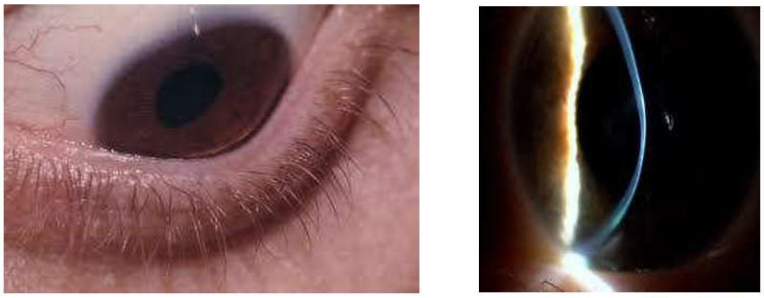

Fig: 21

D. Rizutti sign

a. Illuminated cornea when shined from the temporal side on the nasal part

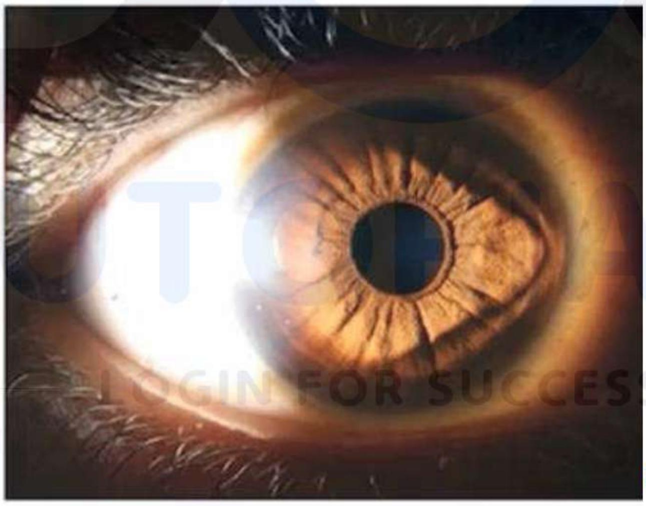

Fig: 22

E.Vogt's striae

a.Vertical Stress lines in the Descemet's Membrane

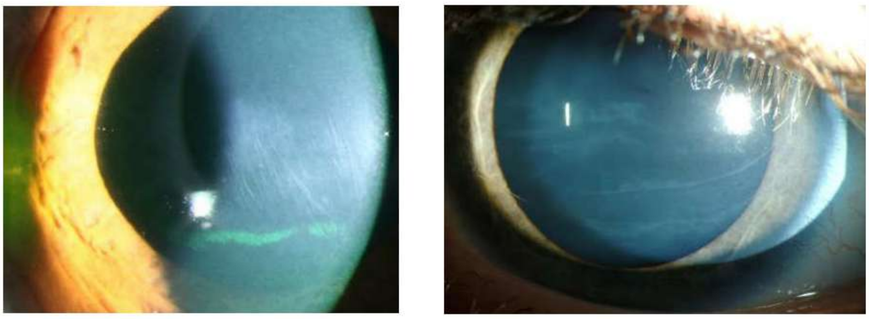

Fig: 23

Note: Horizontal stress lines → Haab's striae

F. Applanation (pulsating mires)
  a. On applanation tonometry
G. Nerves prominent

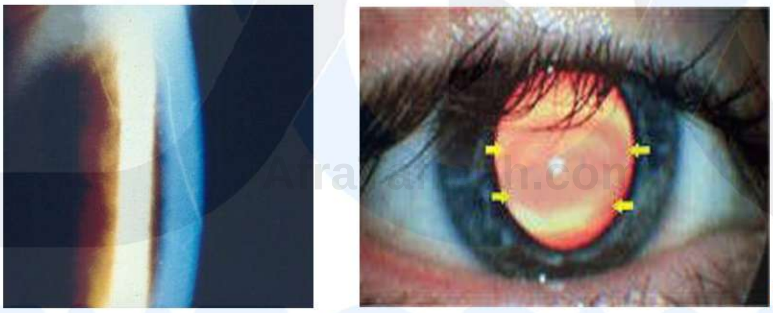

Fig: 24

H. Scissoring reflex
  a. On Retinoscopy
I. Hydrops
  a. Rupture of Descemet's membrane due to acute rise in pressure → Perforation

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Placedo disc 00:28:12

Fig: 25

a. Normal corneal curvature → regular concentric disc

b. Abnormal corneal curvature → abnormal mires

c. Not a diagnostic for Keratoconus

d. Superficial information

Investigation of choice

Pentacam

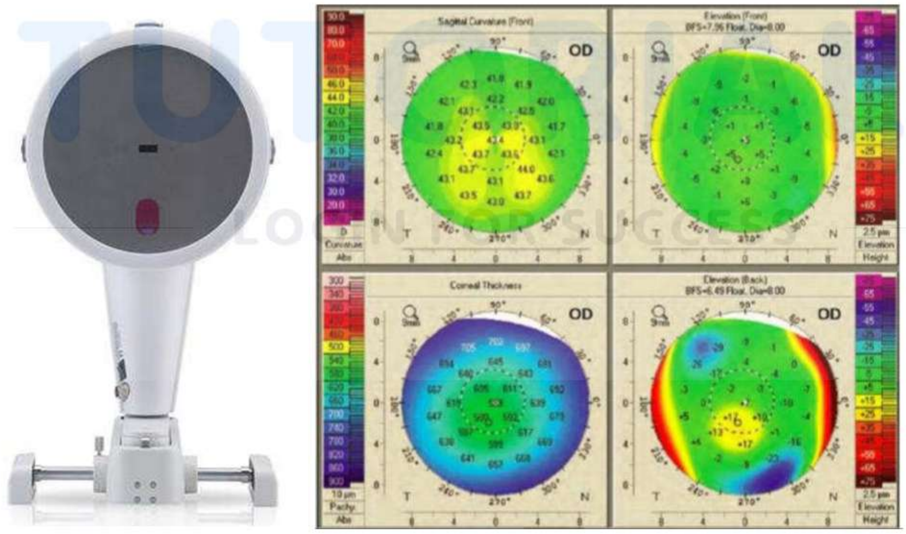

Sagittal Curvature (Front)
OD
Elevation (Front)
OD
Corneal Thickness
OD
Elevation (Back)
OD

Fig: 26

a. Gives topography of cornea

b. Based on Schinfler principles

Interpretation: Green is for Normal and Red is for Thinning (especially in the Centre)

Treatment 00:30:57

A. Replacement of spectacles is just temporary

B.RGP lens (contract lens)

  a. Rigid gas permeable lens

  b. Semisoft

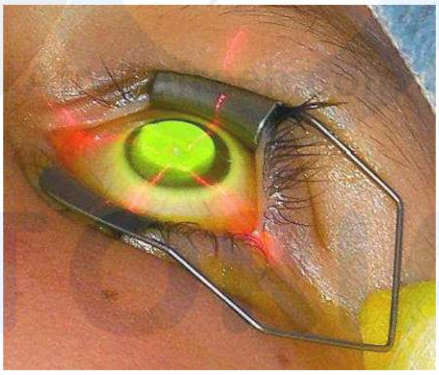
  c. Try to alter the curvature of cornea when applied sufficiently and regularly

C.C3R (Best choice) (TOC)

  a. Corneal collagen cross linkage with riboflavin drops

  b. Vitamin B2 Drops as a de-epithelized cornea followed by exposure to UV Light for 30 mins

  c. Increases cross linkage bonds



c. Fig: 27

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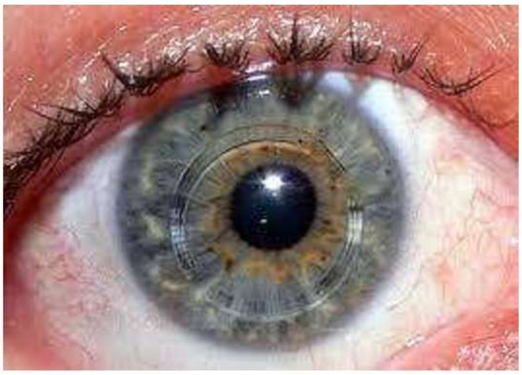
D. INTACS

Fig: 28



E.

a. PMMA crescentic rings in the stroma of cornea
b. Intrastromal corneal ring segment
c. Corrects irregularities of cornea and fattens the cornea
d. Can be used in association with C3R

E. **Pellucid marginal degeneration**

a. Apex and corneal thinning
b. Inferior corneal thinning with a corneal protrusion above the thinnest area
c. Close differential of keratoconus

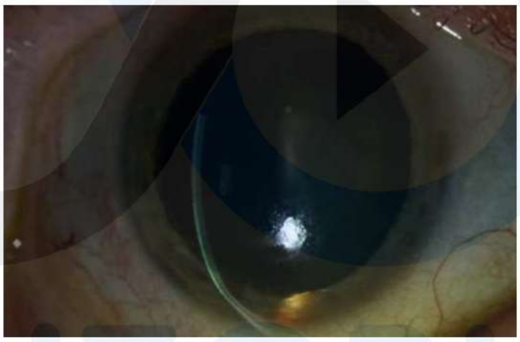


Fig: 29

F. **Keratoglobus**

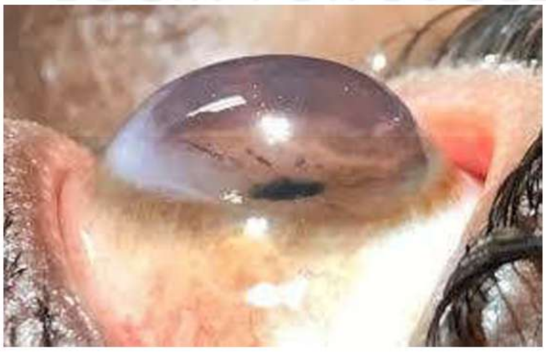


Fig: 30

a. Entirely protruded cornea
b. Can be associated with keratoconus, Lieber's congenital amaurosis, blue sclera

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MISCELLANEOUS TOPICS (CORNEA)

Corneal opacities 00:00:08

Nebular opacity

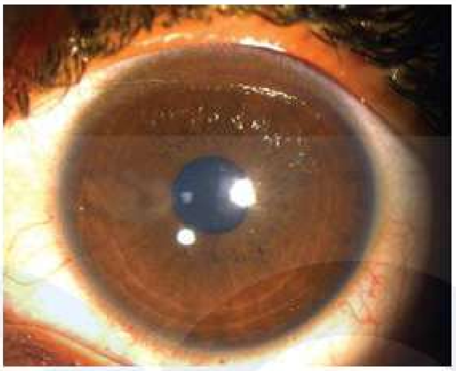

Fig: 1

- Faint, hardly seen with a naked eye
- Seen decrease slit lamp examination only
- Underlying iris is clearly visible
- Stroma (<1/3) affected

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Macular opacity

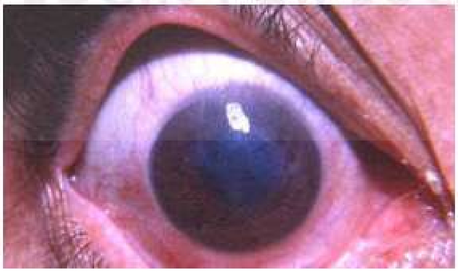

Fig: 2

- Can be seen with naked eye
- < ½ corneal stroma is affected
- Underlying iris details are not very clear

Leucomatous opacity

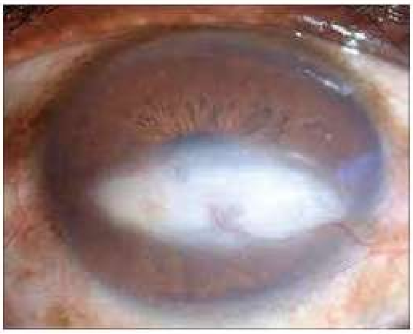

Fig: 3

- Very dense
- > ½ corneal stroma is affected

Treatment options (depending on the depth)

- Change the corneal
- Penetrating/deep anterior lamella keratoplasty
- If it is a non-seeing eye no potential for vision (Corneal tattooing) → cosmetic purpose
- Color of the iris will be given to the cornea
- Leucumatous corneal
- Aniridia +
- India ink is most commonly used
- Platinum and gold salts can also be used

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Photo ophthalmia 00:03:36

- Ultraviolet light exposure
- Multiple superficial corneal erosions (very painful)
- 6-8 hrs. After exposure (late onset)
- Treatment → apply pad/tear supplements /cycloplegic agents

  → Crooks glass is used to prevent; it absorbs UV light

INTRODUCTION (UVEA)

Anatomy of uveal tract 00:00:35

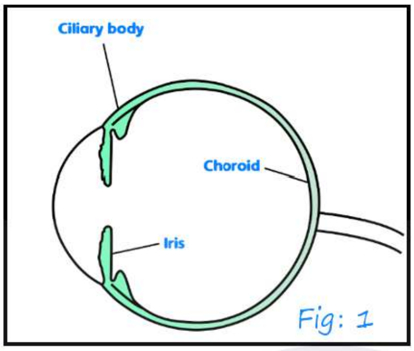

Ciliary body
Choroid
Iris

Fig: 1

- Uveal tract – Middle vascular coat of eyeball
- Outer → Sclera & cornea
- Uveal tract starts anteriorly as iris,
- middle (intermediate) portion – Ciliary body
- Posteriorly – choroid
- Ciliary body has 2 portions

Anteriorly → Pars plicata

Posteriorly → Pars plana

Anatomy of Iris 00:01:30

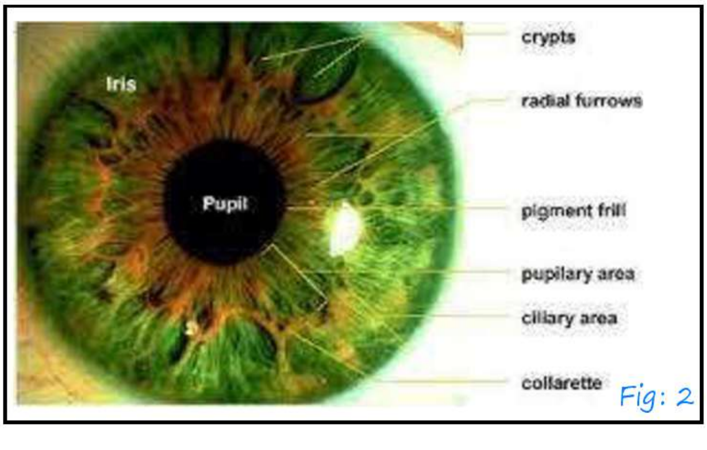
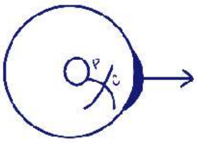

Iris
Pupil
crypts
radial furrows
pigment frill
pupilary area
ciliary area
collarette

Fig: 2



Root of iris

↓

Weakest/Thinnest part

↓

Hence during blunt trauma →
Detachment from root (+)

- Collarets divide iris into 2 zones

Pupillary area	Ciliary area
↓	↓
Towards pupil	Towards cilia

- Collarets denotes attachment of 1° vitreous (Persistent pupillary membrane)
- Depressed weak areas are called crypts are thin area of iris

↓

Site for iridotomy

- Neovascularisation of iris starts at the pupillary border

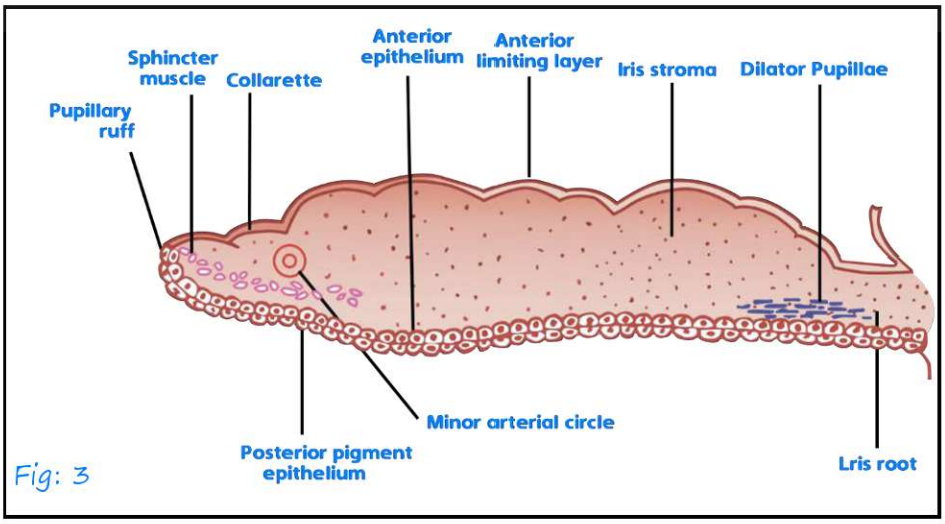

Pupillary ruff
Sphincter muscle
Collarette
Anterior epithelium
Anterior limiting layer
Iris stroma
Dilator Pupillae
Minor arterial circle
Posterior pigment epithelium
Lris root

Fig: 3

Layers of Iris

- 1st layers → Outer most layer
- Anterior limiting layer
  - Pigments like melanocytes
  - Langerhans
- 2nd layer → Stroma
  - Maximum thickness
  - Also has melanocytes
  - All muscle is present in stroma
- Colour of iris is due to number of melanocytes melanin cells in the anterior limiting layer
- 3rd layer
  - Anterior epithelium
  - Continuation of pigmented ciliary body
  - Continuation of retinal pigment epithelium of retina
- 4th layer
  - Inner most layer
  - Posterior pigment epithelium
  - Continuation of non-pigmentation epithelium of ciliary body
  - Continuation of neurosensory retina

Layers of ciliary body 00:07:45

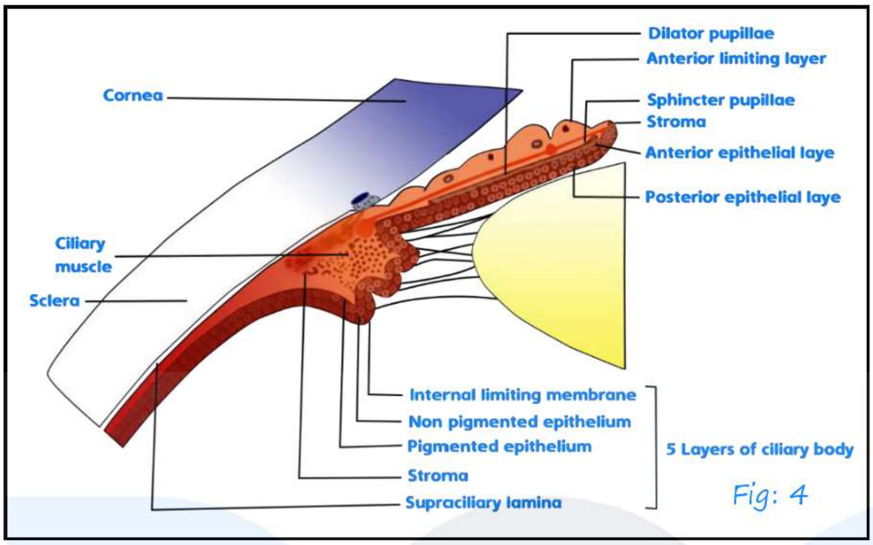

Dilator pupillae
Anterior limiting layer
Cornea
Sphincter pupillae
Stroma
Anterior epithelial laye
Posterior epithelial laye
Ciliary muscle
Sclera
Internal limiting membrane
Non pigmented epithelium
Pigmented epithelium
Stroma
Supraciliary lamina
5 Layers of ciliary body

Fig: 4

- 5 layers
  - Internal limiting membrane
  - Non pigmented epithelium
  - Stroma
  - Supraciliary lamina

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Choroid 00:08:45

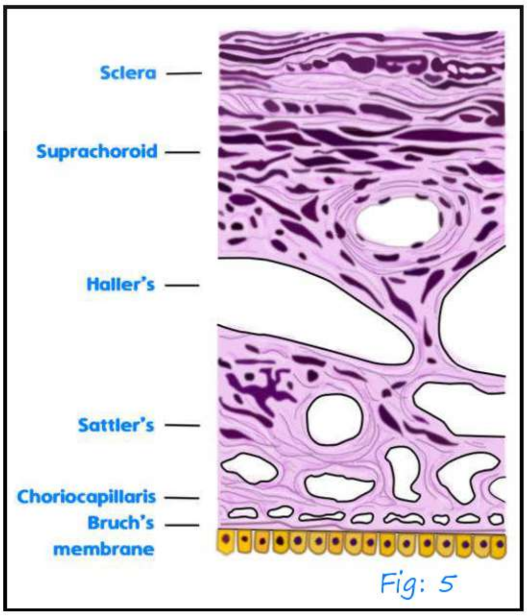
Sclera
Suprachoroid
Haller's
Sattler's
Choriocapillaris
Bruch's membrane

Fig: 5

4layers

i. Anterior → Suprachoroidal lamina

ii. Stroma → Has 2 layers → Haller's (Bigger blood vessels) & Sattler's

iii. Choriocapillaris

iv. Bruch's membrane

Blood supply of eye 00:10:13

- Main artery → Ophthalmic artery

Central retinal artery

Choroidal artery

Muscular artery
↓
Fermius anterior ciliary arteries
↓
2 muscular arteries for each recti except lateral rectus

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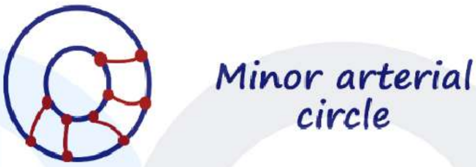
Choroidal artery carry

Short posterior artery
↓
20 in number
↓
Around optic nerve

Long posterior ciliary artery
↓
2 in number
↓
Travels in space and goes anteriorly to supply the cornea sclera

- Anterior ciliary artery and long posterior ciliary artery make plexus around the root of iris → Major arterial circle
- Clinical importance → Blunt trauma shows hyphema → Blood comes from major blood source
- Branches coming at major arterial circle anastomose at pupillary margin



UVEITIS DEFINITION & TYPES

- Uveitis → Inflammation of uveal tract
- Based on anatomical location uveitis can be

  i. Anterior → Iritis, Iridocyclitis

  ii. Intermediate → Pars planitis & hyalitis

  iii. Posterior

  - Choroiditis
  - Retinochoroiditis

  iv. Panuveitis → Inflammation of whole of uveal tract (1,2,3 together)

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According to clinical course

- Acute – Sudden onset inflammation with limited duration (<3months)
- Chronic – Persistent inflammation with prompt release in less than 3months after stopping treatment
- Recurrent → Repeated episodes separated by at least 3months of inactivity without treatment

ANTERIOR UVEITIS – CLINICAL FEATURES, MANAGEMENT & COMPLICATIONS

Anterior uveitis

- Iritis/Iridocyclitis

  Most common cause – Idiopathic
- HLA B27 related arthropathy
- Ankylosing spondylitis → Back ache
- Reiters disease → H/O infectious diarrhoea
- Inflammatory bowel disease
- Psoriatic arthritis

Reiters disease

- Conjunctivitis
- Arthritis
- Urethritis
- Inflammatory bowel disease → Ulcerative colitis & crohn's disease
- JRA → juvenile rheumatoid arthritis
- Usually pauciarticular <5 joint involvement → Most commonly cause uveitis
- Most common cause of uveitis in children – JRA
- JRA – Most common cause of band shaped keratopathy in children

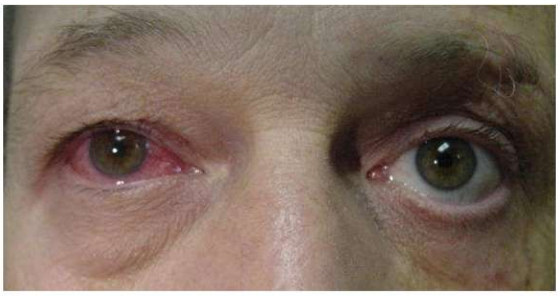
Infectious causes

- Granulomatous
  - TB
  - Sarcoidosis
  - Syphilis
  - Hansen's disease
  - Lens induced uveitis
  - VKH syndrome
  - Herpes

Connective tissue disorders

- PAN
- Wegener's
- SLE
- Skin disorders
- Behcet disease
- Fuch's uveitis
- Posner schlossman syndrome

Symptoms 00:06:00



Right anterior Uveitis Fig: 1

- Pain
- Decreased vision
- Redness → circumcorneal
- Photophobia → More prominent

Causes of decreased vision in uveitis

- Turbidity of aqueous
- Cystoid macular edema
- Induced myopia-ciliary muscle contraction

Slit lamp examination

- Cells – In AC → Small dots moving → PMN → Cell is the marker of activity
- Flare – In AC → Accumulation or leakage of lipids from inflamed capillaries
- Gives turbidity to aqueous
- Marker of severity

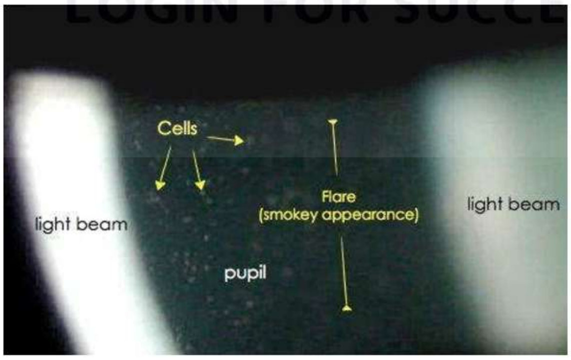

Cells
light beam
Flare
(smokey appearance)
light beam
pupil

Fig: 2

Magnified view of slit → 1mm x 1mm

SUN grading 00:11:32

Grade	No of AC cells
0	<1
0.5+	1-5
1+	6-15
2+	16-25
3+	26-50
4+	>50

Grade	Anterior chamber flare
0	Note
1+	Faint
2+	Moderate (Iris and lens details clear)
3+	Marked (Iris and lens details hazy)
4+	Intense (Fibrin or plastic aqueous)

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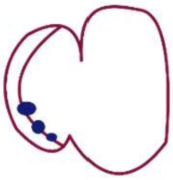
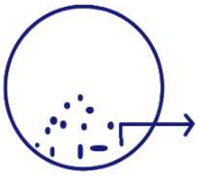
Grading of vitreous haze

Score	Description	Clinical findings
0	Nil	None
0.5+	Trace	None
1	Minimal	Posterior pole clearly visible
2	Mild	Posterior pole details slightly
3	Moderate	Posterior pole details very hazy
4	Marked	Posterior pole details barely visible
5	Severe	Fundus details non visible

Keratic precipitates (KP) 00:12:58

- settle down inferiorly on back of cornea





- Art's triangle – Configuration of arrangement of keratic precipitates settle down when you look from back
- KP → Indication of etiology

Granulomatous → Mutton fat KP's

Non granulomatous → Thin flat KP's

- Stellate KP's: Herpes, Toxoplasmosis, Fuch
- Pigmented KP's – Healed uveitis
- Flare – Last thing to persist

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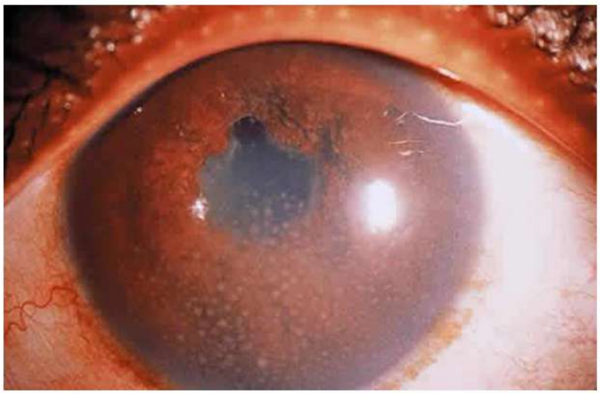
Changes in IRIS

- Boggy iris



Irregular Pupil Fig: 3

- Pupil will be irregular → due to Synechiae & small → due to spasm of sphincter muscle
- IRIS and lens are joined together → Synechiae formation

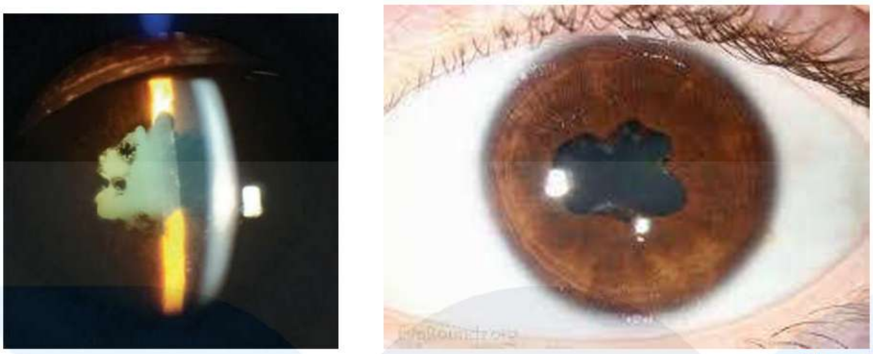
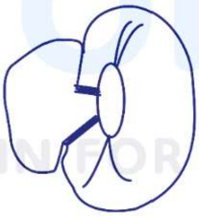


Fig: 4

Synechiae

- Anterior → Adhesion between cornea and iris
- Posterior → Adhesion of iris to lens
- Peripheral → Adhesion at angle itself



↓

These Adhesions are 360° @ pupillary margin

↓

Ring synechiae

↓

Seclusio pupillae

↓

Bending of iris → Iris bombe

↓

2° angle closure glaucoma

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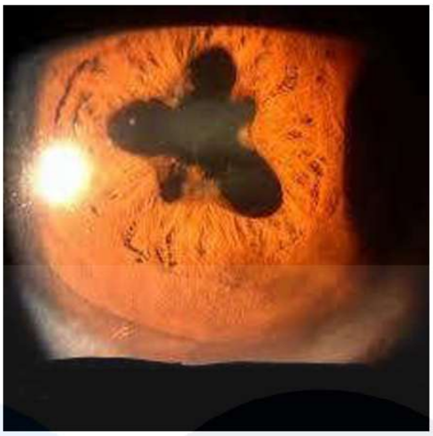
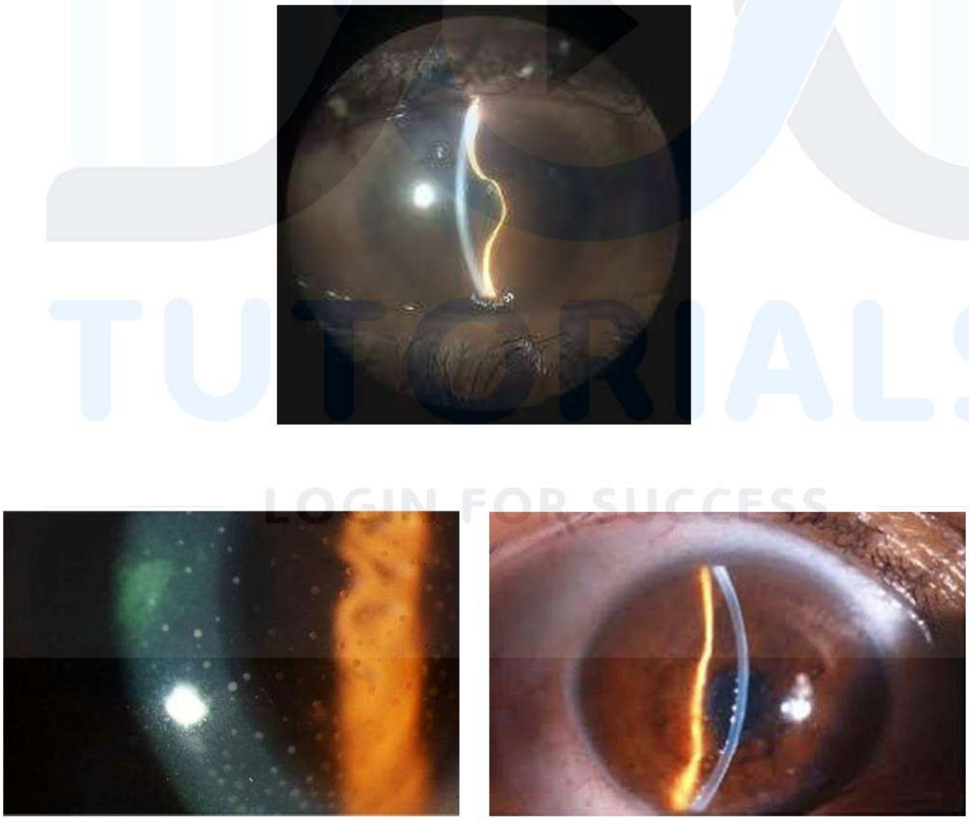
- Inflammatory area over iris

  ↓

  Occlusio pupillae



Festooned pupil Fig: 5



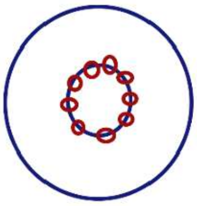
Keratic Precipitates Fig: 6

- Nodules in iris

  ↓

  3 types

a. Koeppe's nodules – generally present at the pupillary margin



- These nodules are made up of macrophages epithelioid cells

b. Busacca's nodules – generally seen @ base

↓

More pathognomic for granulomatous uveitis

c. Berlin nodules – typically found in angle

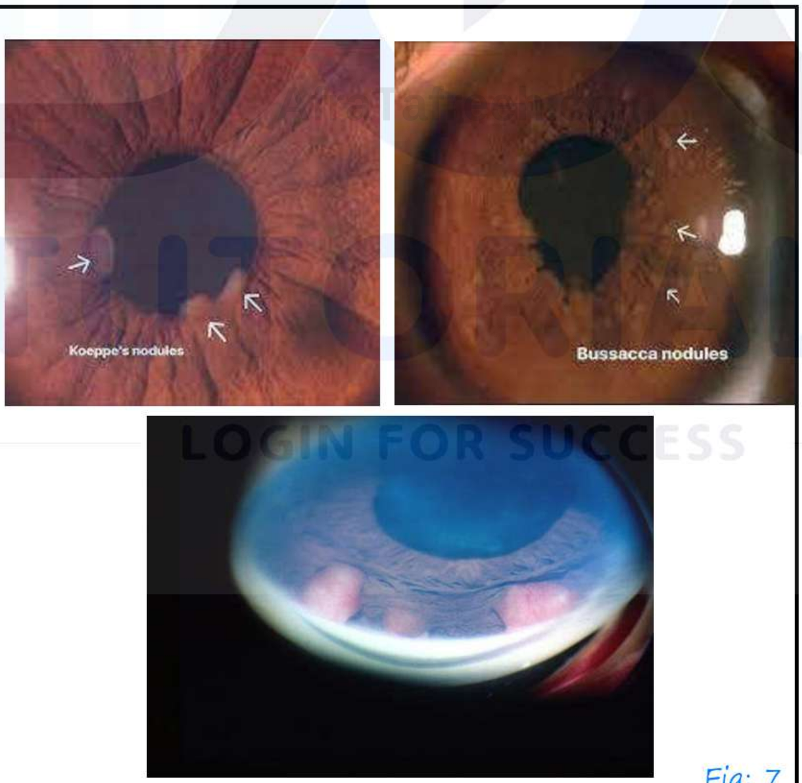


Fig: 7

Hypopyon → pus in anterior chamber

- Cold mobile hypopyon → Behcet's disease → white eye

Treatment 00:26:12

1.Steroids – Topical → DOC

2.Cycloplegic mydriatic → Atropine, homatropine, tropicamide

Initially atropine → short acting

Roles

a. Relieves pain (most important)

↓

By reducing the ciliary muscle spasm

b. Break synechiae

c. Prevent synechiae formation

d. Increase vascularity

3.To decrease IOP → β blockers/oral acetazolamide

Pilocarpine ⎤
Pg analogue ⎦ C/I

↓

S/E → Inflammation

Complications 00:31:00

1.Complicated cataract → most common complication in chronic uveitis

2.Glaucoma → most common complication

2° OAG → d/t inflammation

2° ACG → d/t Iris bombe synechiae

3.Band shaped keratopathy

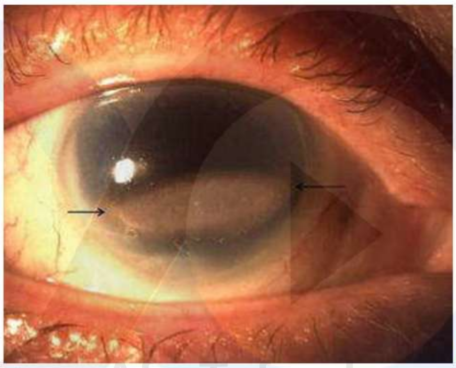


Fig: 8

- Accumulation of calcium in a band shaped form

4.Cystoid macular edema → most common cause of decreased vision in uveitis

5.Phthisis bulbi

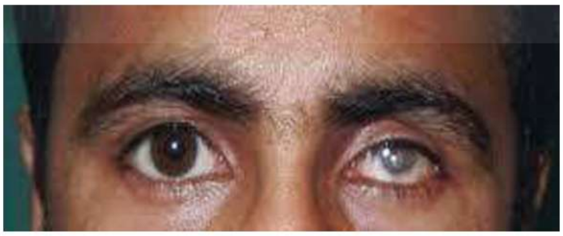


Fig: 9

- 7's-Soft, shrunken, sore, square, shapeless, sightless, structureless

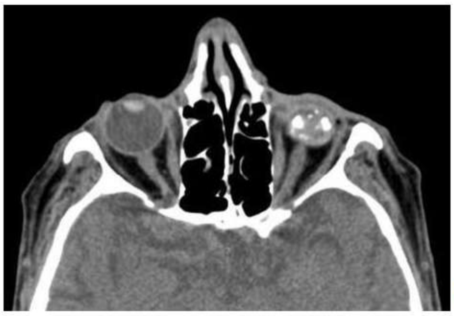


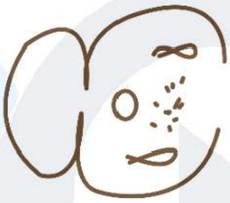
Fig: 10

- Foci of calcium deposits ossification in the sclera, cornea, lens, retina and optic nerve

INTERMEDIATE UVEITIS

- Pass planitis: - idiopathic cases of intermediate uveitis (most common)
  - H/o TB, sarcoidosis, Lyme's disease syphilis, multiple sclerosis

Symptoms 00:01:25



- Complaint of - floaters
  - Metamorphosis
  - Decreased vision
- Floaters – small black objects which are seen as shadow floating
- Increased cells in vitreous cavity

Signs 00:04:00

- Indirect ophthalmoscope

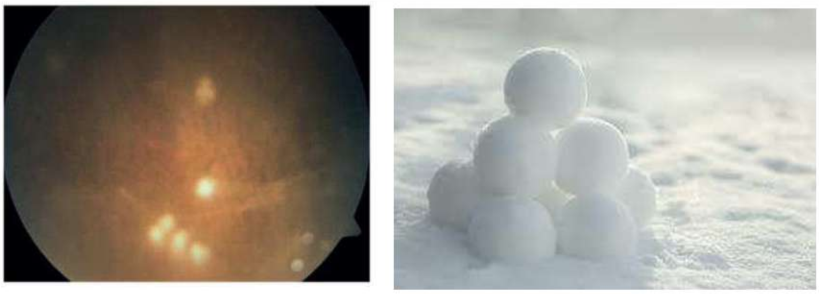


Fig: 1

i. Snowball → small exudation in anterior vitreous

→ more or less seen in inferior part

ii. Snow banking – pathognomonic

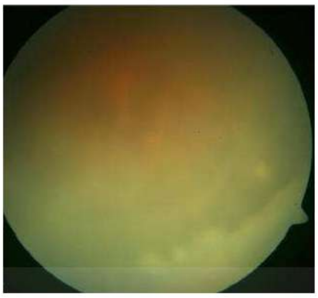


Fig: 2

Plaque – whole of pars plana has exudation

iii. Candle wax dripping

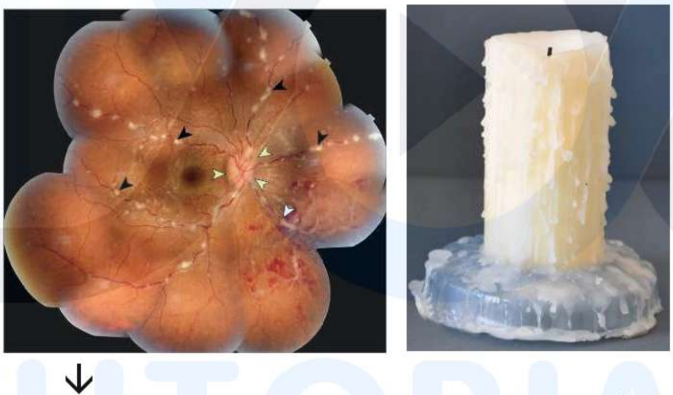


Fig: 3

Perivascular sheathing → sarcoidosis

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Treatment 00:06:25

- 4 step approach of KAPLAN

i. Topical steroids

↓

ii. Steroids periocular (posterior subtenons injection)

↓

iii. Immunomodulatory agents

↓

iv. PPV (pars plana vitrectomy) + laser photocoagulation

POSTERIOR UVEITIS

- Inflammation of choroid- chorioretinitis , also can involve retina – chorioretinitis

Etiology

- Toxoplasmosis → most common cause
- Sarcoidosis
- Tuberculosis
- Ocular syphilis
- Herpes
- HIV
- VKH syndrome
- Toxocariasis

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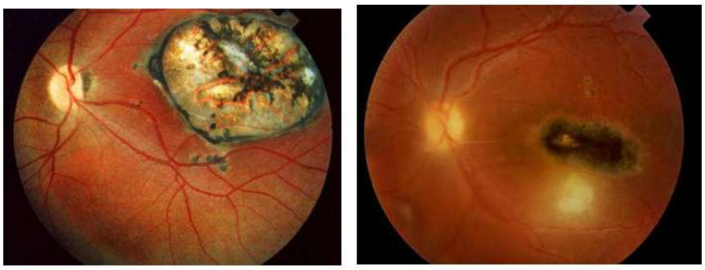
Toxoplasmosis 00:01:20

- Most common cause of infectious retinochoroiditis in immunocompetent patients
- Most common etiology → uncooked meat, meat infected with cat's faces

  - Cat- definitive host
  - Human – (intermediate Host)
- Involvement of posterior polar regions in the form of macular scar
- Most common cases

  ↓

  Congenital cases

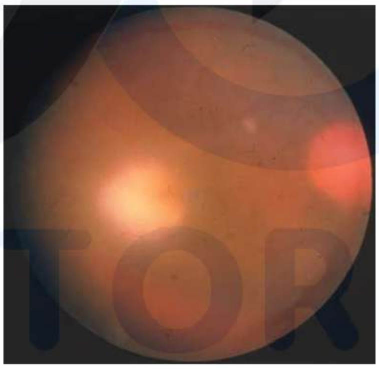


Toxoplasma scar satellite lesion Fig: 1

- Clinical feature – spill over anterior uveitis
  - Satellite lesion
  - Old macular scar
  - In Active choroiditis

    ↓

    Head light in fog appearance



Active choroiditis Fig: 2

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Light in fog appearance Fig: 3

- Fog – vitritis
- Head light → granuloma

Treatment 00:05:55

- Triple therapy → P – pyrimethamine
  S – sulphadiazine
  C – corticosteroids
  +
  Clindamycin
  — Quadruple therapy

Toxocariasis 00:06:45

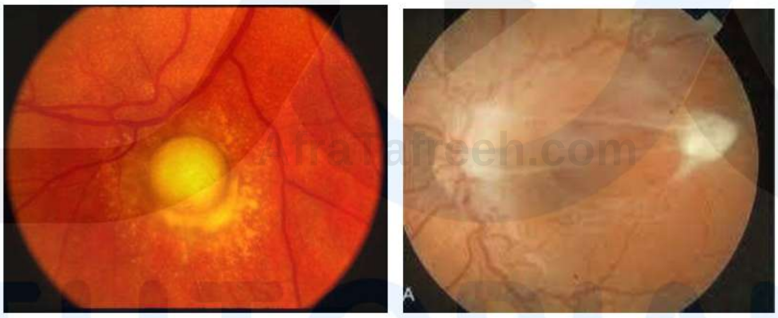

Fig: 4

- Round worm infection
- Presentation – posterior pole granuloma or a peripheral granuloma

↓

Macular dragging can occur

→ endophthalmitis

→ leucocoria can also be seen

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HIV retinopathy 00:08:18

- Most common presentation of ocular HIV → HIV retinopathy

↓

Cotton wool spots

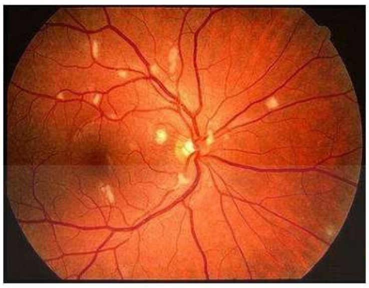


Fig: 5

- CD4 < 50 cells / microliter → CMV can cause infections

↓

CMV retinitis

CMV reaches retina in many forms

→ most common – hematogenous route

→ infects endothelium of capillaries

→ immune complex deposition

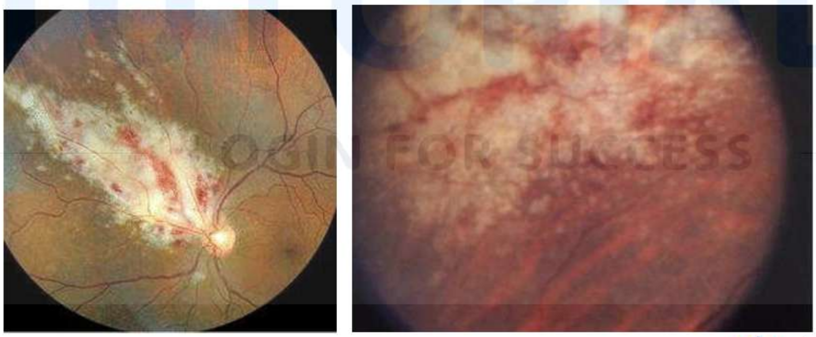


Fig: 6

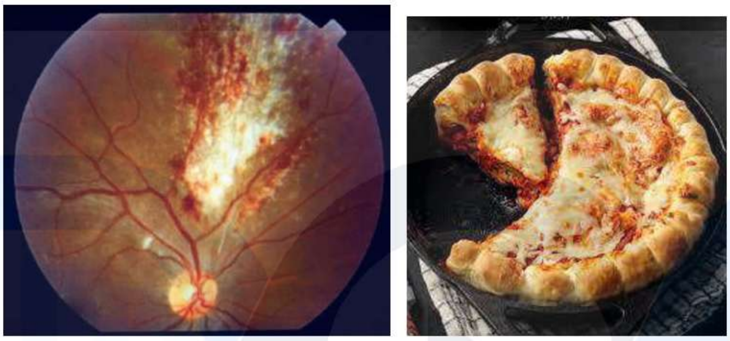
- Indolent form of CMV retinitis

↓

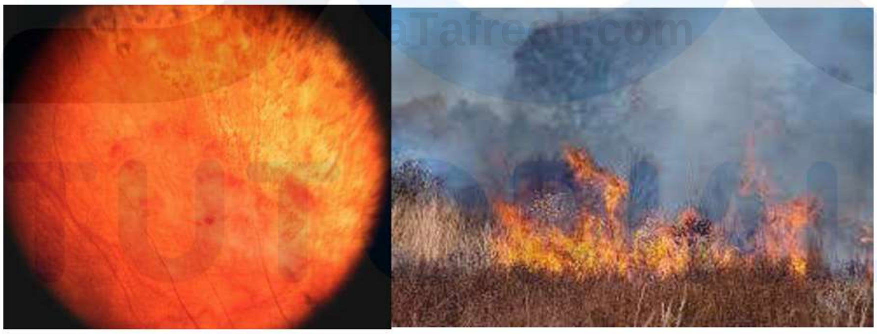
  - Slow progression
  - Starts from periphery and moves towards center
  - White opalescent
  - Granular appearance

  - Creates atrophic regions
  - Less hemorrhages

- Pizza pie appearance /cotton cheese with ketchup – large areas of retinal necrosis and hemorrhage along vascular arcades. The advancing edge these lesions usually spread contiguously



Fulminant retinitis Fig: 7



↓ Fig: 8

Brush fire extenstion can also be present

- Frosted branch angitis is mostly vasculitis

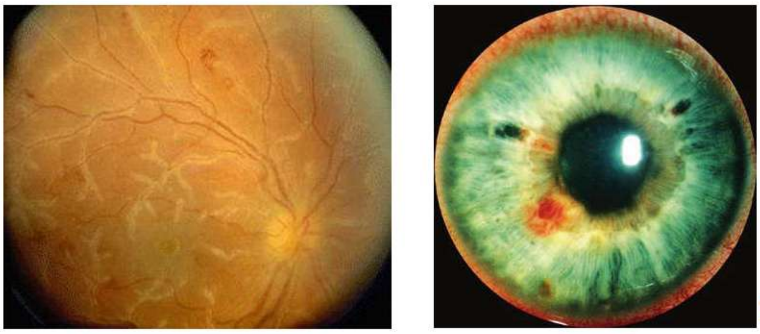


Fig: 9

↓

Iris Roseola

- Occular syphilis → vascularised nodule
- Iris pearls-

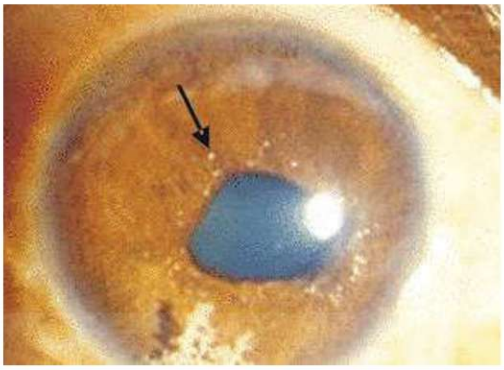


Fig: 10

↓

Small white pin point lesions along puplillary margins

↓

Accumulaiton of lepra bacill

Occular sacrcoidosis

- Anterior uveitis
- IU, posterior, chorodiitis, posterior uveitis
- Periphlebits, perivenous sheathing
- Candle wax dripping sign
- Choroiditis
- Snowball-string pearls apperance
- Retinal granuloma-lander's sign
- Optic disc edema
- Most common presentaiton – anterior uveitis

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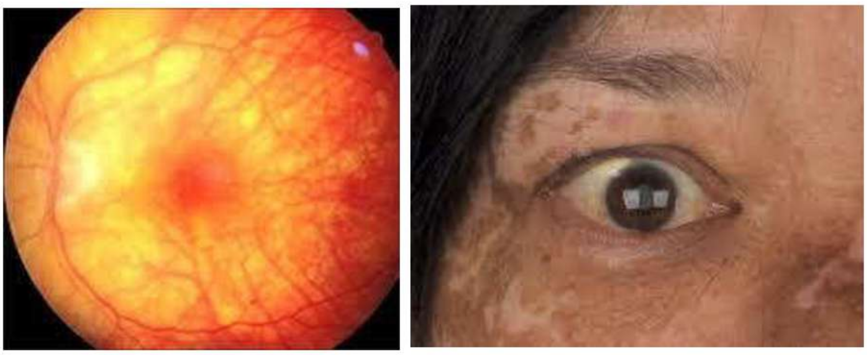
PANUVEITIS, VKH SYNDROME & SYMATHETIC OPHTHALMIA

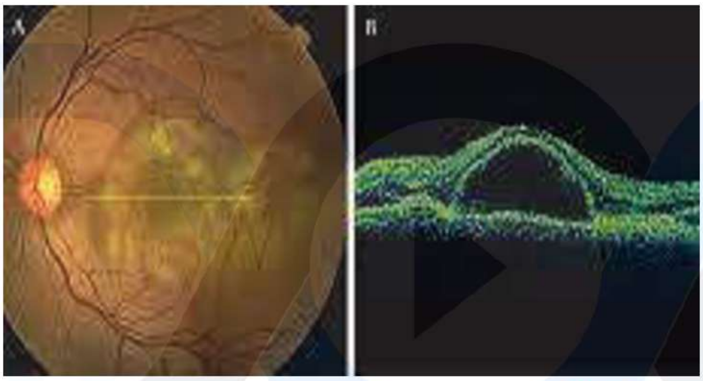
- Panuveitis – inflammation of all parts of uveal tract
- Causes – Toxoplasmosis
    - Toxocariasis
    - TB
    - Sarcoidosis's
    - Lyme's disease
    - VHK syndrome
    - Sympathetic ophthalmia

VHK syndrome 00:00:56

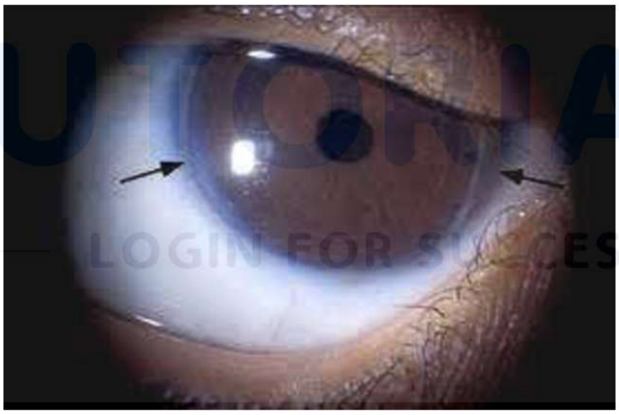
- Vogt-Koyanagi-Harada syndrome
- Mostly Idiopathic
- Multisystemic involvement in tissues which contains melanocytes
- Systems involved → eyes
    - → skin
    - → ENT
    - → meninges
- Eyes →Granulomatous panuveitis, exudative retinal detachment, perilimbal vitiligo, sunset glow fundus
- Skin→ vitiligo, alopecia
- ENT→ tinnitus, vertigo
- Meninges→ meningitis, CSF pleocytosis



RPE atrophy vitiligo Fig: 1



RD Fig: 2



Sugiura Sign_Perilimbal vitiligo Fig: 3

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MISCELLANEOUS TOPICS

Fuch hetero chronic iridocyclitis

- Heterochromia – change in color of iris
- Decrease in vision due to cataract
- Minimal inflammation → painless gradual loss of vision mostly after development of cataract

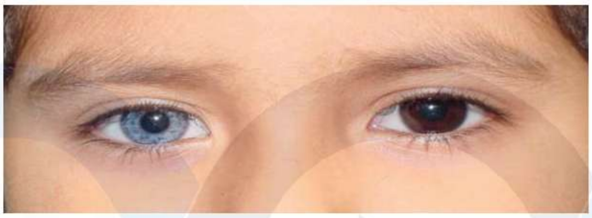
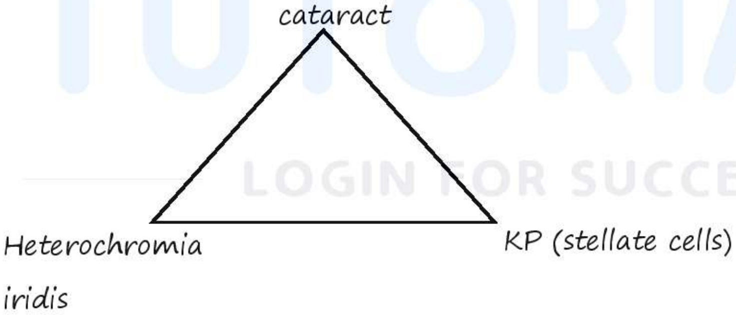
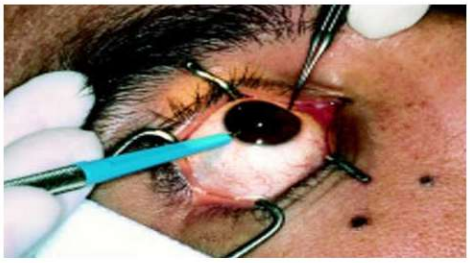


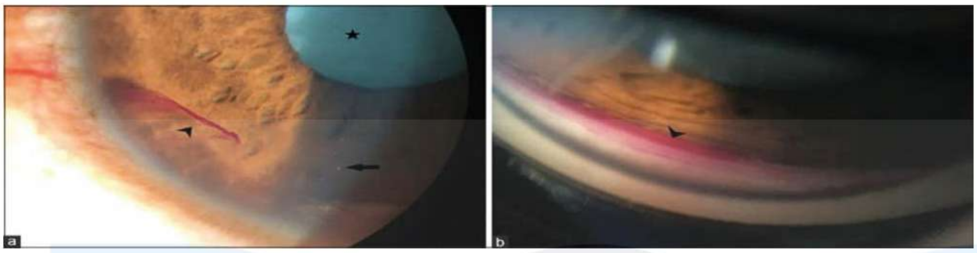
Fig: 1

- Never develop synechiae
- KP'S – diffuse, stellate
- Good prognosis after doing cataract surgery
- TRIAD (CHIK)


cataract
Heterochromia iridis
KP (stellate cells)



paracentesis



↓

Amsler's sign: bleeding in AC due to new vessels

Fig: 2

- Other causes of Heterochromia
    - Congenital Horne's syndrome
    - Waardenburg syndrome
    - Oculodermal melanosis
    - Diffuse iris melanoma
    - Siderosis
    - Extensive rubeosis
    - Herpes simplex uveitis
    - PG analogues
    - Nevus of Ota

Fuch Heterochromic iridocyclitis	Acute anterior uveitis
Asymptomatic	Pain, redness, photophobia, blurred vision
Minimal reaction, diffuse KPs	Cells ++, flare ++ Kp inferiorly
No synechiae	Synechiae+
Doesn't require steroids	Steroids are drugs of choice

Posner schlossman syndrome /glaucomatocyclitic crisis

- Young → males > females
  ↓
  Recurrent high IOP
- Minimal AC reaction – scanty KPs
- CMV can be associated
- Trabeculitis – primarily inflammation of Trabecular meshwork
- Control IOP
- steroids+

White dot syndrome & HLA Associations

MEWDS	Birdshot chorioretinopathy	Serpiginous choroiditis	APMPPE	PIC
Young, U/L	Old ages 5-7 decade Females HLA = A29	4 to 6 decade, males BL HLA B8	Rare B/L 3 to 5 Decade	Young Myopic women B/L
Preceding viral illness	Chronic recurrent	Recurrent chronic	Follow flue like illness	Absent vitritis
Numerous small white ill defined	Multiple yellow white lesion all around	Punched out wavy lesions	Large cream-colored placoid lesion	Small multiple yellow white spots inner choroids
Macula spared	Macula plus peripheral	Vitritis, uveitis, RPE choroidal atrophy +	RPE involved	Fuzzy borders
Excellent prognosis	Vitritis ++	Poor prognosis		Chronic lesions atrophy, pigmented

MEWDS (Multiple excrescent white dot syndrome)

- Young, U/L
- Preceding viral illness
- Numerous small white ill defined
- Macula spared
- Excellent prognosis

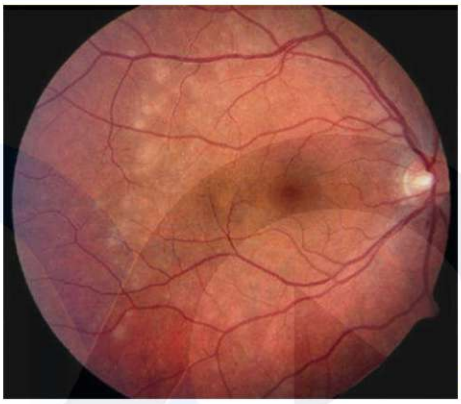


Fig: 3

Bird shot chorioretinopathy

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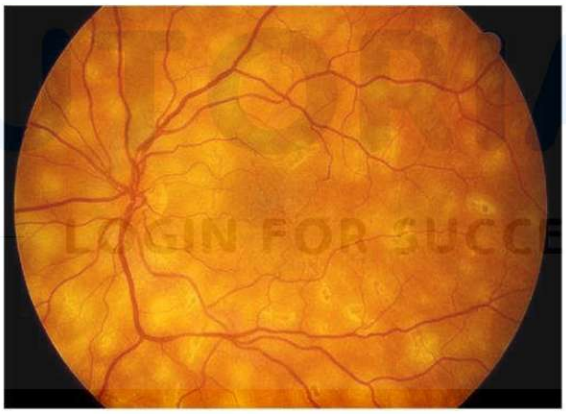


Fig: 4

- Old ages 5-7 decades females
- HLA – A29
- Chronic recurrent
- Multiple yellow white lesions all around
- Macular plus peripheral
- Vitritis ++

Serpiginous choroiditis

- 4-6 the decade, males B:
- HLA B7
- Recurrent chronic
- Punched out wavy lesions
- Vitritis, uveitis, RPE choroidal atrophy+
- Poor prognosis
- Large lesions

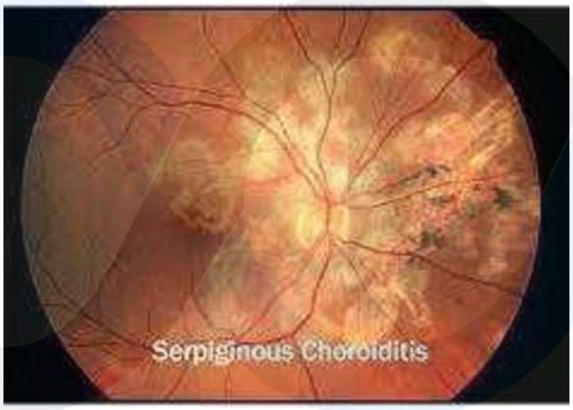
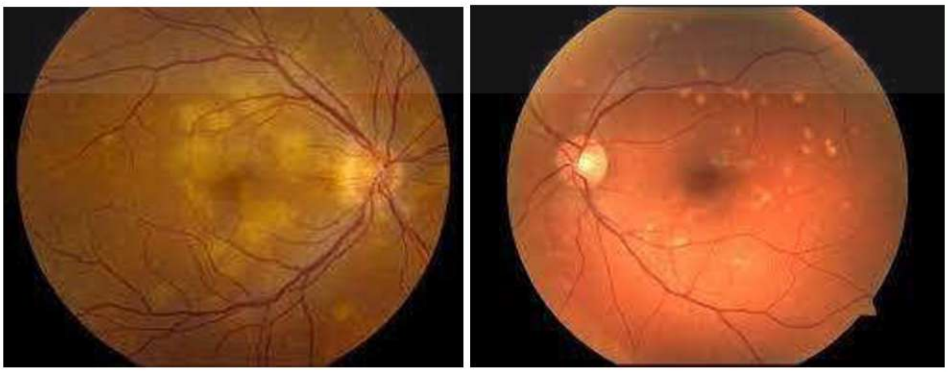


Fig: 5

APMPPE

- Acute posterior multifocal pigmentary placoid epitheliopathy


AMPPE PIC Fig: 6

- PIC- punctate inner choroidopathy
- Young myopic females

HLA Association

HLA	Disorders
HLA A29 HLA B7	Birdshot chorioretinopathy Serpiginous choroiditis
HLA B27	Arthropathies non granulomatous Uveitis
HLA B5, HLA B51	Behcet disease
HLA DR4	VKH< sympathetic ophthalmia
HLA B8	Sarcoidosis

INTRODUCTION AND ANATOMY PHYSIOLOGY OF LENS

- Lens is avascular
  Biconvex ⎤ structure situated in patellar fossa
  Clear
  Crystalline

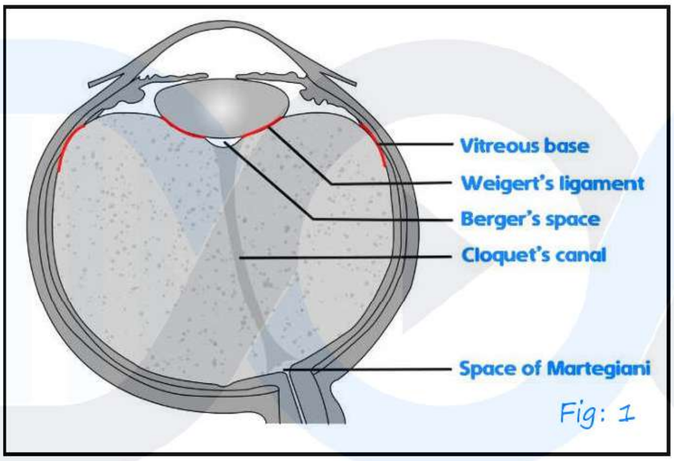

Vitreous base
Weigert's ligament
Berger's space
Cloquet's canal
Space of Martegiani

Fig: 1

- Posterior part of lens is in contact with anterior part of vitreous in circular form
  ↓
  Wiegert's ligament (Hyaloid capsular ligament)
- Empty space just behind posterior capsule and anterior phase of vitreous → Berger's space
- From Berger space to optic nerve
  ↓
  Cloquet's canal
  ↓
  Also called hyaloid canal

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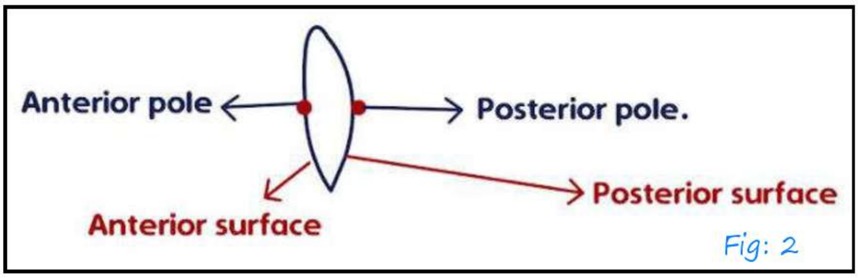
Anterior pole
Posterior pole.
Posterior surface
Anterior surface

Fig: 2

Vitreous base – area near ora serrata where vitreous and retina are stronger

- Lens is biconvex

↓↓

But, relatively anterior surface of lens is relatively flatter compared to posterior surface

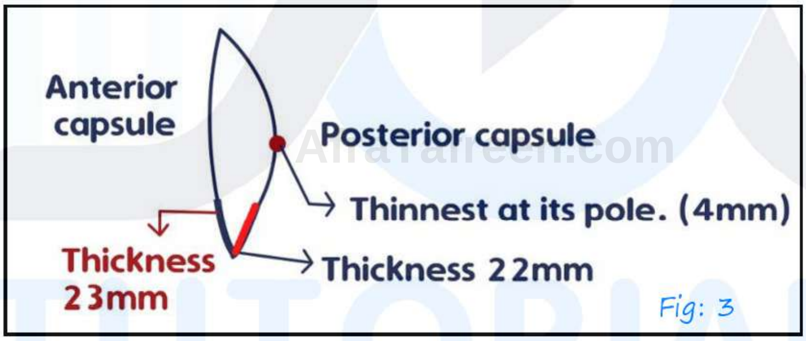
Anterior capsule
Posterior capsule
Thinnest at its pole. (4mm)
Thickness 23mm
Thickness 22mm

Fig: 3

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The most prominent part in anterior surface is anterior pole and in posterior Surface is posterior pole

Anterior diameter is 10 mm

- Posterior diameter is 6 mm
- Thickness
  - At birth: - 2.5 mm – 3 mm

    ↓↓

    Elderly: - 4.5 mm – 5 mm
  - Approximately 0.05 mm increase in thickness occurs every year

- Lens is avascular
  - Source of nourishment – aqueous humor (vitreous humor)

Metabolism

- Primarily anaerobic
- HMP shunt
- Krebs cycle
- Glycolysis

Refractive index

- In periphery 1.39 ⎤ Differential refractive index
  Centre 1.42 ⎦

Layers of lens

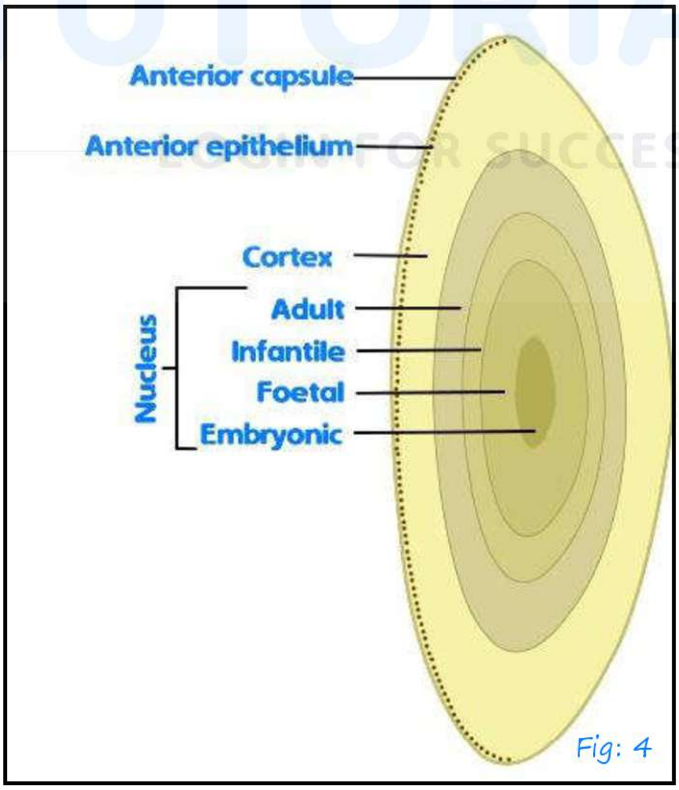

Anterior capsule
Anterior epithelium
Cortex
Nucleus
Adult
Infantile
Foetal
Embryonic

Fig: 4

- 4 layers
- Outer side – capsule
- Just inside capsule – anterior epithelium

↓

Cortex

↓

Nucleus

- Nucleus is divided into 4 zones

Innermost – embryonic

↓

Fetal

↓

Infantile

↓

Adult

1.Capsule

- Mainly made of type IV collagen
- Thickest basement in the body
- Highly elastic in young
- No elastin
- Anterior capsule is secreted continuously by the anterior epithelial cells
- Posterior capsule – also secreted by post epithelium but not continuously
- Gradually thickening of anterior capsule as we grow
- Posterior capsule thickness remains same or decreases
- Posterior capsule is thinnest at pole, (4 mm)

- Capsule is thinnest at post pole and thickest at pre-equatorial region

2.Epithelium

- Anterior epithelium
- Posterior epithelium
- Epithelium is utilized in formation of this fibers

↓

Absent in adults

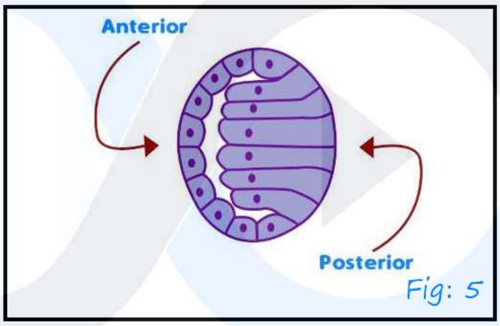


Fig: 5

TUTORIALS
LOGIC FOR SUCCESS

- During intrauterine life, lens is a spherical structure

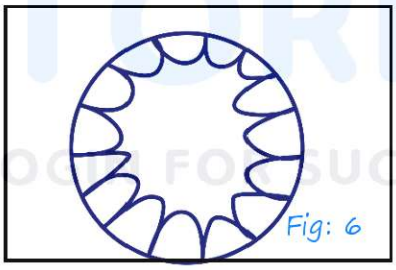


Fig: 6

- During gestation, 1st month → posterior epithelial cells elongated → and finally used up
- Embryonic nucleus → first formed lens fibers

↓↓ ↓

Source → Post epithelium 0.3 month of gestation

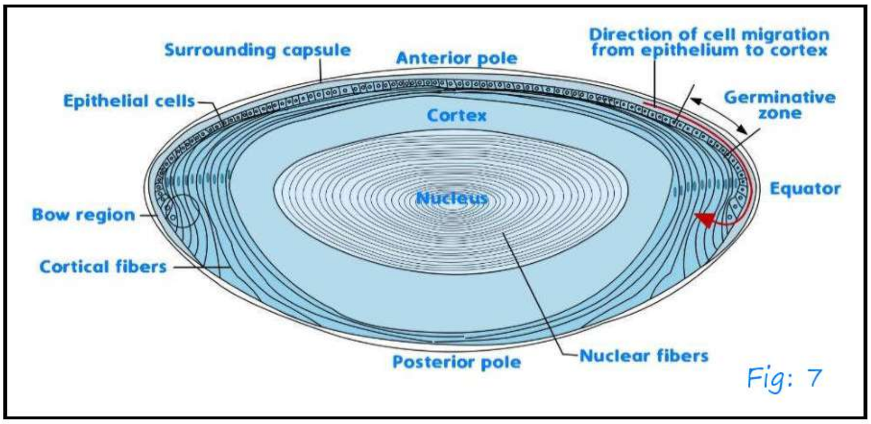

Surrounding capsule
Anterior pole
Direction of cell migration from epithelium to cortex
Epithelial cells
Cortex
Germinative zone
Equator
Nucleus
Bow region
Cortical fibers
Posterior pole
Nuclear fibers

Fig: 7

Ant. Epithelium → Central cells – cuboidal → 'A' cells

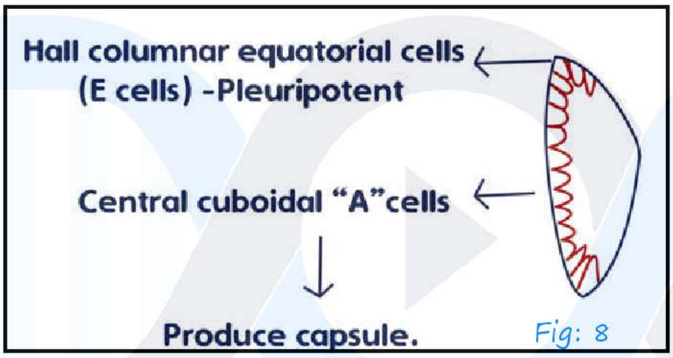

Hall columnar equatorial cells (E cells) -Pleuripotent
Central cuboidal "A"cells
Produce capsule.

Fig: 8

TUTORIALS
LEARN FOR SUCCESS

Peripheral cells – Pre equatorial
↓
Columnar
↓
'E' cells
↓
Continue to produce secondary lens fibres

3.Cortex

- Loosely arranged lens fibres
- Newest lens fibres
- Source is 'E' cells

4.Nucleus → 4 zones

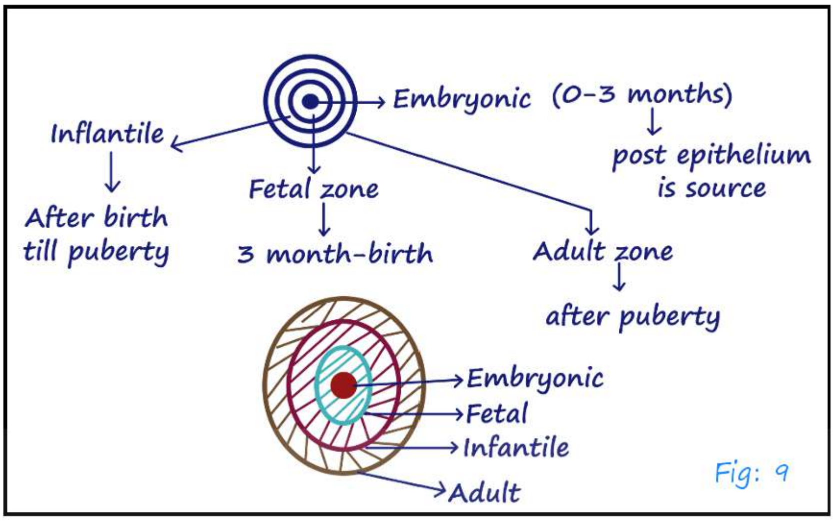

Embryonic (0-3 months)
↓
post epithelium is source
Inflantile
↓
After birth till puberty
Fetal zone
↓
3 month-birth
Adult zone
↓
after puberty
Embryonic
Fetal
Infantile
Adult

Fig: 9

- From E cells, there is continuous production of new cells, goes posteriorly and later pushed back anteriorly, nuclei will be lost

↓↓

Nuclear Bow would be created

↓↓

Bow region

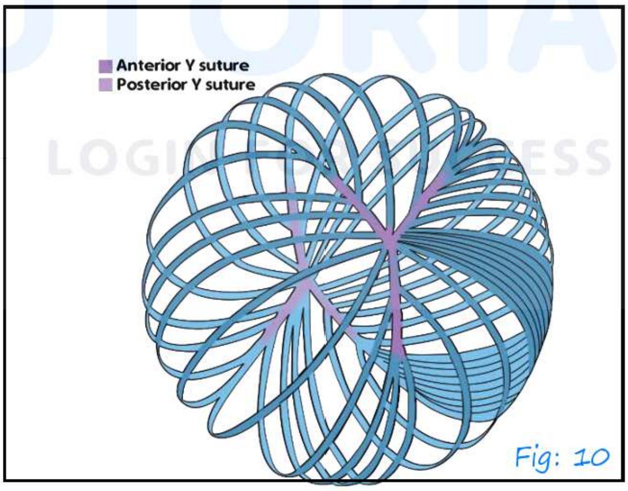

Anterior Y suture
Posterior Y suture

Fig: 10

- Y shaped sutures
  - Anteriorly – Erect Y → apical cells meet
  - Posteriorly – inverted Y – basal cells meet

- Meeting of lens fibres at same plane at pole

Zonules and accommodation

- Lens is suspended by zonules all around
- In each eye, there are about 70-80 zonules

Scanning electrogram of zonules

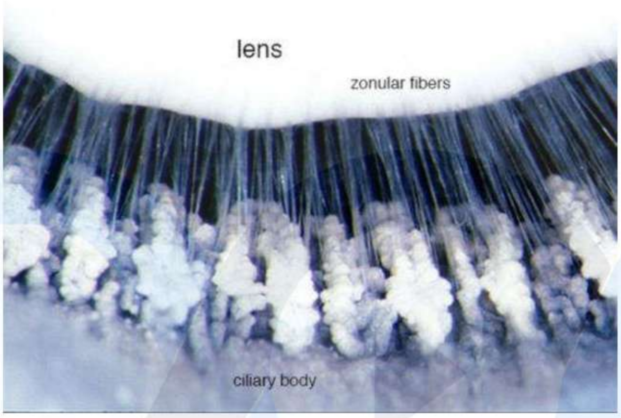

lens
zonular fibers
ciliary body

Fig: 11

↓

Arrangement of zonules

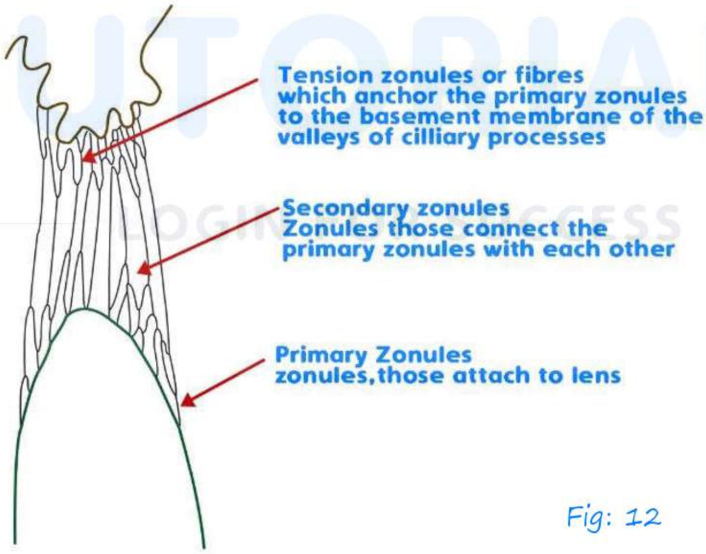

Tension zonules or fibres which anchor the primary zonules to the basement membrane of the valleys of cilliary processes
Secondary zonules Zonules those connect the primary zonules with each other
Primary Zonules zonules,those attach to lens

Fig: 12

- These zonules are mainly coming from pars plana and go to pars plicata

↓

Zonular fork

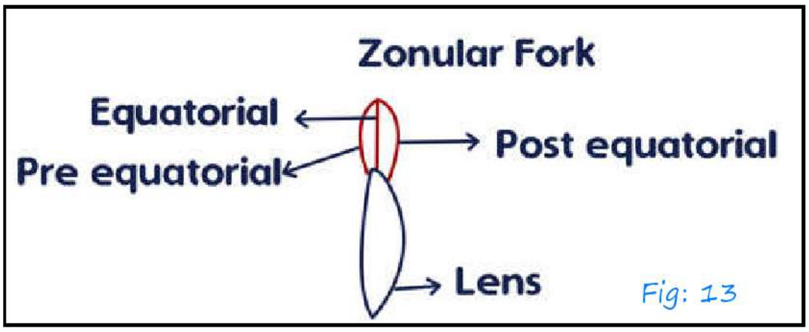

Zonular Fork
Equatorial
Pre equatorial
Post equatorial
Lens

Fig: 13

- Few are arranged posteriorly, few in midline and few on pre-equatorial zone
- Tension zonules – most important zonules for accommodation

Accommodation

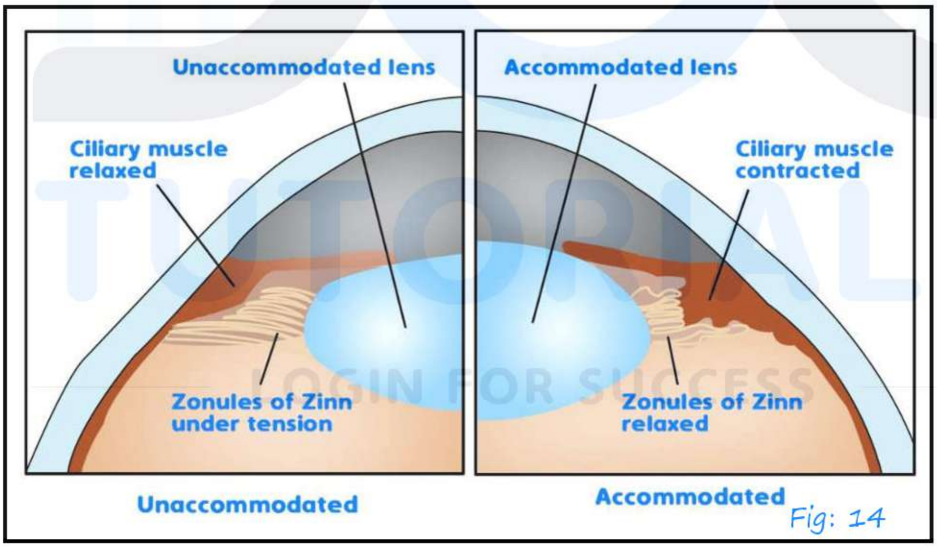

Unaccommodated lens
Ciliary muscle relaxed
Zonules of Zinn under tension
Unaccommodated
Accommodated lens
Ciliary muscle contracted
Zonules of Zinn relaxed
Accommodated

Fig: 14

- When looking at far gaze
  ↓↓
  Lens are stretched
  ↓↓
  Zonules are also stretched
  ↓↓
  Quickly when I shift the gaze to near object

↓↓

Accommodation

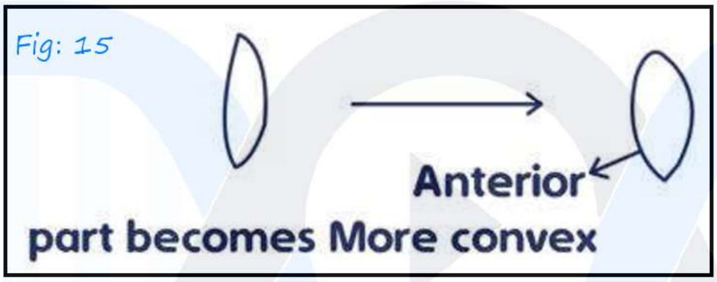
- Accommodation → changes produced in the eye to look at a near object
- Changes in eye

1.Contraction of ciliary m/s

2.Relaxation of zonules

3.Lens become more convex

Fig: 15



Anterior part becomes More convex

- Anterior part becomes more convex
- Post part remains same

4.Miosis

5.Lens iris diaphragm shifts slightly forward

6.Convergence

- Near reflex trad

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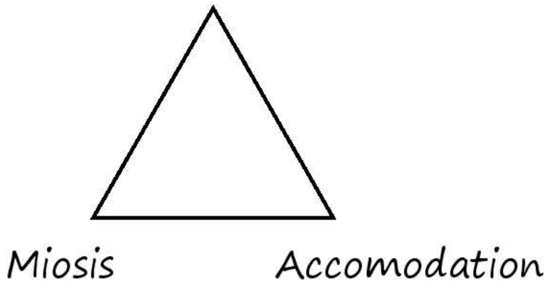
CAM

↓

Convergence



Antioxidants and proteins in lens

- Antioxidants
  - Vit. C
  - Vit. E
  - Glutathione
  - Superoxide dismutase
  - Catalase
  - Peroxidase
- Water content → 65%
- Proteins → 35%   VI% lipids and carbohydrates

Proteins in lens

Water Soluble

↓

Crystallins

↓

- α – heaviest heat shock proteins
- β – maximum in number
- γ – present in fetal life

Insoluble

↓

- vimentin
- aquaporins

↓

- Membrane intrinsic protein (MIP -26)

↓

Hydration in lens

Cataractogenesis

DOI TUTORIALS LOGIN FOR SUCCESS

MANAGEMENT OF CATARACT

Management of cataract

- Check for best corrected visual acuity

  ↓

  Rule Out glaucoma

  Check IOP and posterior segment
- USG – B Scan → tells us about posterior segment when mature cataract is present
- Macular function tests

Indications for surgeries

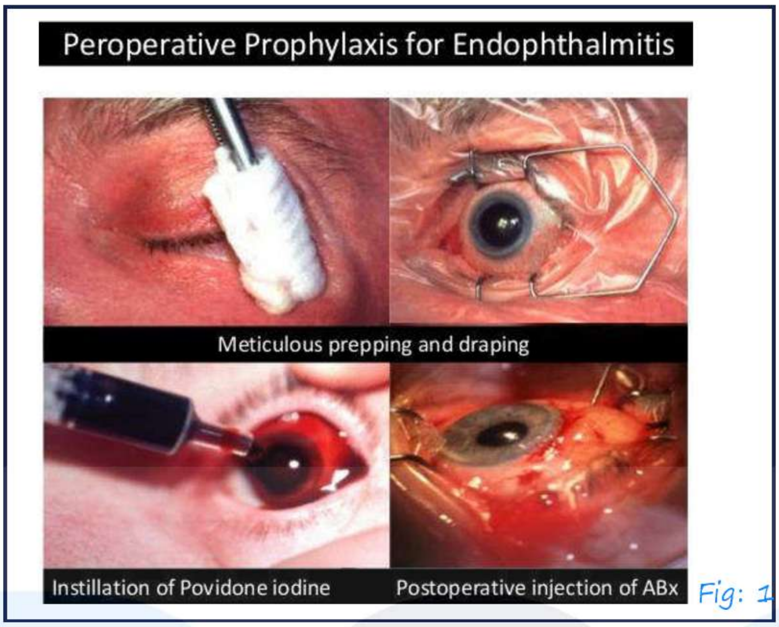
- Even if vision is 6/6 but patient is symptomatic

  ↓

  - Blurred vision
  - Glare

Patient preparation 00:04:04

- Eye drops
  - NSAIDS
    - Prevent CME
    - Prevent intraoperative miosis
  - Pre-op antibiotics ±



Peroperative Prophylaxis for Endophthalmitis

Meticulous prepping and draping

Instillation of Povidone iodine

Postoperative injection of ABx

Fig: 1

- Best prophylaxis for endophthalmitis

↓↓

Standard povidone solution one minute before making incision

- Anesthesia: - Topical → Proparacaine drop

Work up 00:07:45

- IOL Power check up → Keratometry

↓

A-Scan

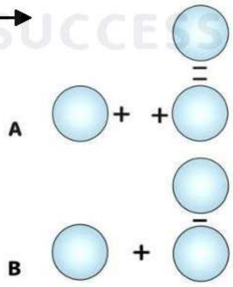
↓

Amplitude scan

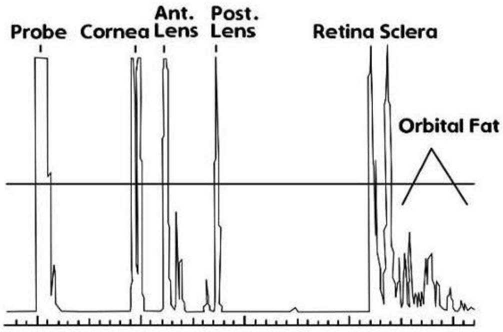
↓

Bared on principle of ultrasound

↓



Based on first purkinje image



↓

Axial length determined
from A-scan

IOL power calculation →SRK II formula (Sanders, Retzlaff, kraff)

- IOL power → A – 2.5L – 0.9K (Eye length 22-24 mm)

  A – Constant

  K – Average keratometry reading

  L – Axial length
- For long eyes → SRK - T formula
- For short eyes → Hoffer Q formula
- Post lasik eye – Haigis formula

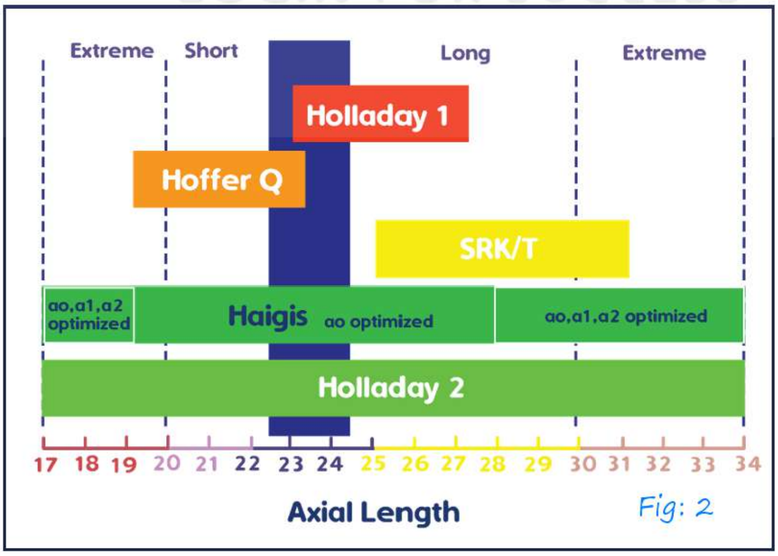


Fig: 2

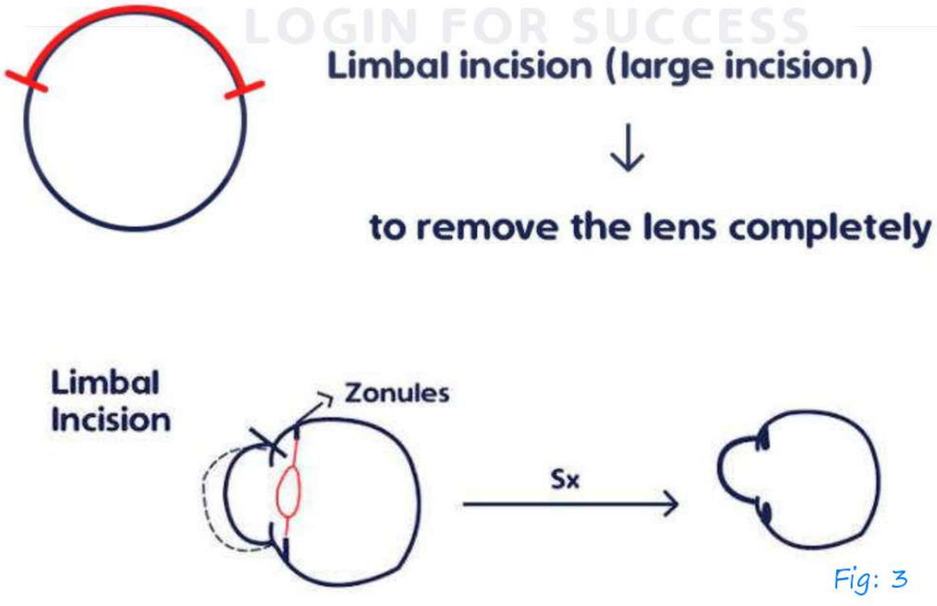
- Holladay 2→ best for all range of eye length

Holladay 2 formula (1988)

- Accurate estimation of the ELPo (Effective lens position)
- Calculates → Axial length
  Central corneal power (K)
  Anterior chamber depth
  Lens thickness measurement
  Limbal white to white measurement
  Age of the patient
  Previous refraction of the patient

Surgery for cataract 00:13:55

Intracapsular cataract extraction 00:14:23



Limbal incision (large incision)
↓
to remove the lens completely

Fig: 3

Complete removal of lens with its capsule

Surgeon used to do incision over superior limbus

↓

Huge incision made and whole of lens with capsule taken out

↓

Zonules are torn

- Anterior chamber IOL is the only option

Problem

- No place for IOL
- Early PVD → Rhegmatogenous retinal detachment (RRD)
- This surgery is almost obsolete
- only indication for ICCE → zonular dehiscence > 180°

Extracapsular cataract extraction 00:18:27

- Only nucleus is taken out
- Cortex aspirated – IOL placed in capsular bag
- Small incision 10 'O' clock – 2 'O' clock

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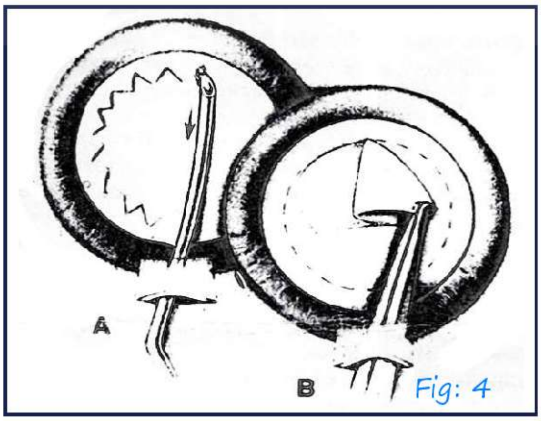


Fig: 4

- Can opener capsulotomy

- CCC- continuous curvilinear capsulorehxis

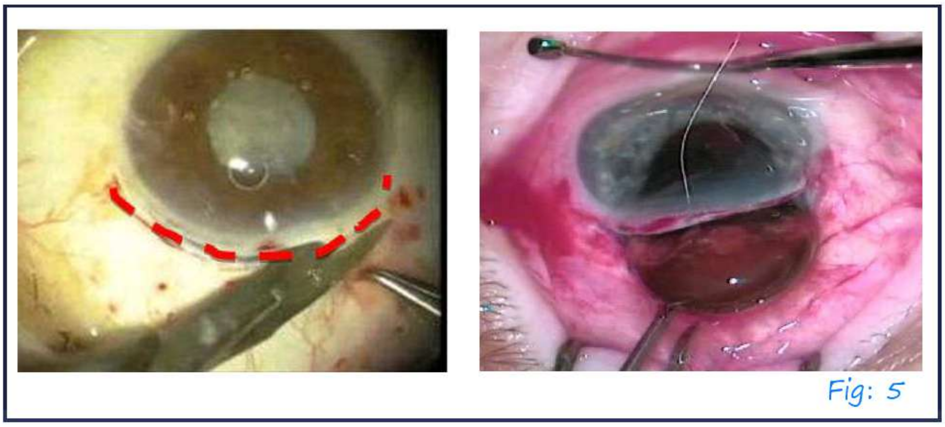


Fig: 5

Advantages

- less chances of RRD
- IOL in capsular bag

Disadvantages

- Astigmation

Small incision cataract surgery (SICS) 00:22:45

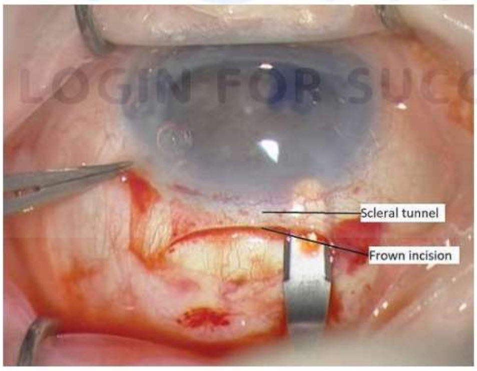
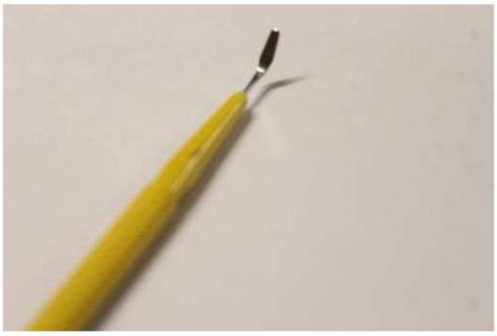

Scleral tunnel
Frown incision

Fig: 6

- Frown shaped incision on sclera



Crescent knife Fig: 7

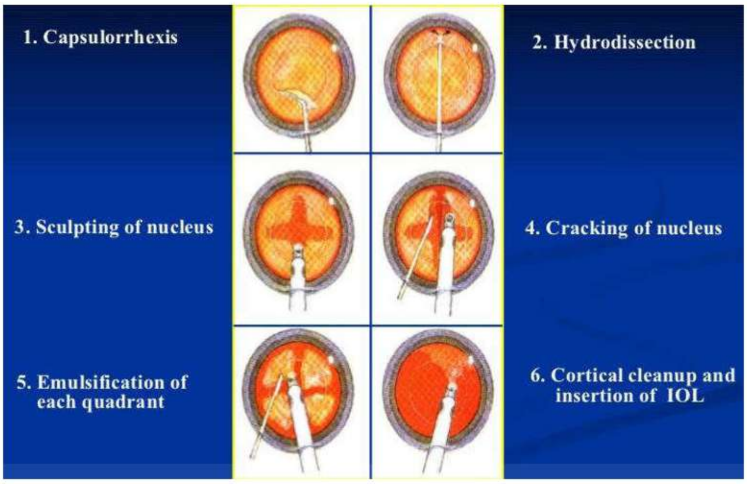
- Tunnel is created though crescent knife from sclera to anterior chamber
- Incision is of 5.5 mm in size
- Chances of minimal astigmatism

Phacoemulsification 00:24:30

AfraTafreeh.com TUTORIALS LOGIN FOR SUCCESS

- Charles David Kelman American ophthalmologist surgeon, inventor, jazz musician, entertainer, and Broadway producer. Known as the father of phacoemulsification.
- 1st phaco in 1962

Fig: 8


1. Capsulorrhexis
2. Hydrodissection
3. Sculpting of nucleus
4. Cracking of nucleus
5. Emulsification of each quadrant
6. Cortical cleanup and insertion of IOL

Phacoemulsification Fig: 9

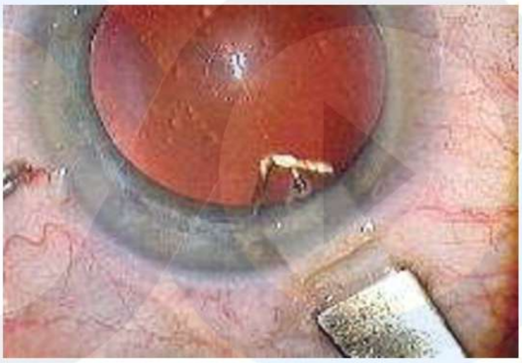


Fig: 10

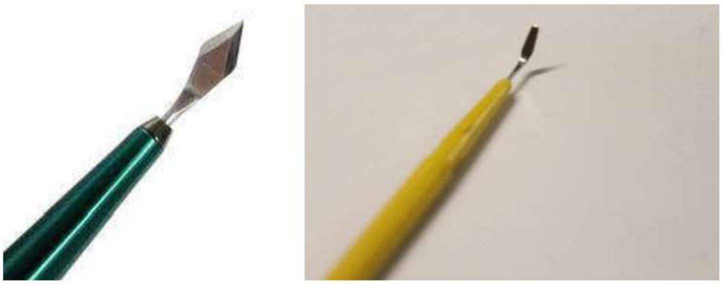
- Step – 1 → clear corneal incision → self sealing

  ↓

  3.2 mm size → standard

  Change size to < 1.8 mm – micro incisional cataract surgery

  - < 1 mm – phaconet

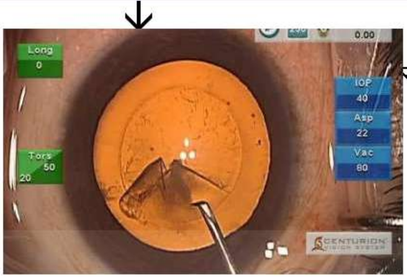


Keratome Fig: 11

- Keratome → used for making corneal incisions

- Step 2 → continuous curvilinear capsulorhexis

↓


Long
0
Tors
50
20
0.00
IOP
40
Asp
22
Vac
80
CENTURION VISION SYSTEM

→ done by cystitome

→ 26 g needle

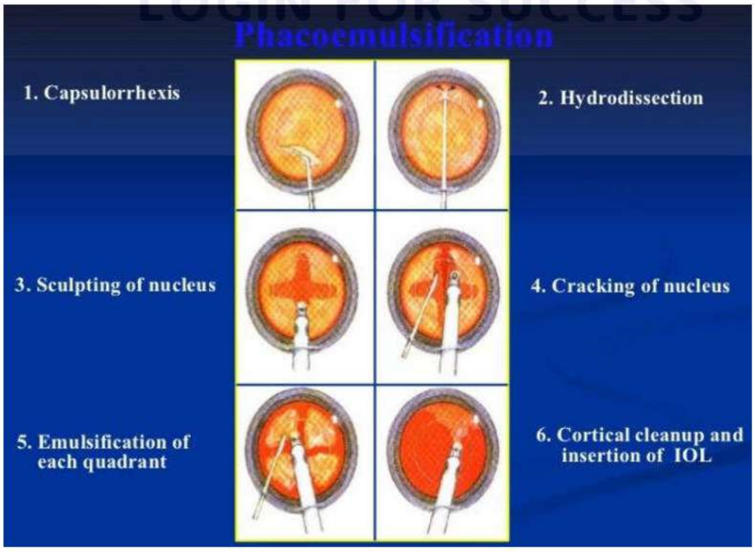
- Size around 5.5 mm
- Prevents extension of radial tears

Fig: 12

Fig: 13

- Blue color dye used to stain mature cataract

DOCTUTORIALS
LOGIN FOR SUCCESS



Phacoemulsification

1. Capsulorrhexis

2. Hydrodissection

3. Sculpting of nucleus

4. Cracking of nucleus

5. Emulsification of each quadrant

6. Cortical cleanup and insertion of IOL

↓

H

Fig: 14

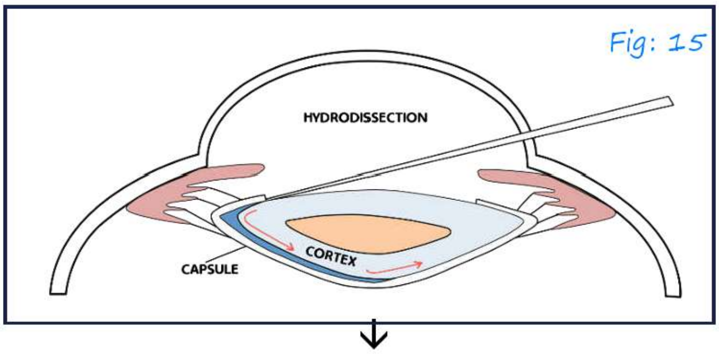

HYDRODISSECTION
CORTEX
CAPSULE

Fig: 15

↓

Hydro dissection

↓

Done from main port

↓

RL/NS is injected

- Separates capsule from cortex

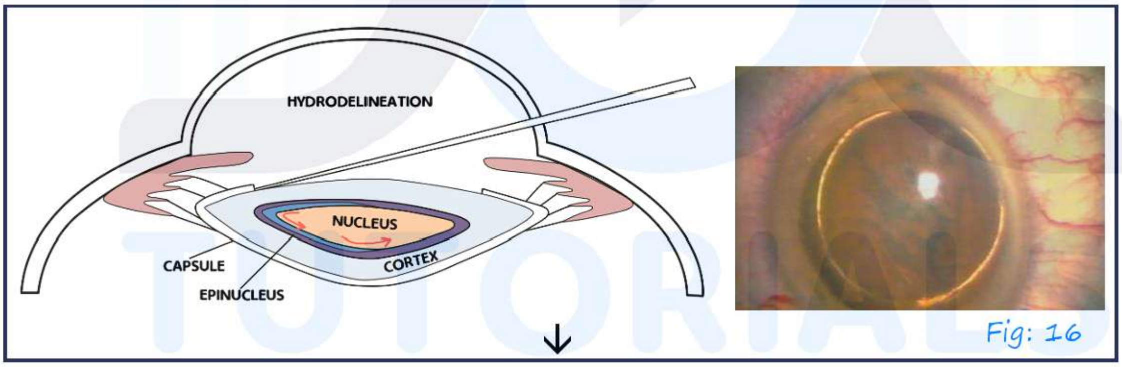

HYDRODELINEATION
NUCLEUS
CORTEX
CAPSULE
EPINUCLEUS

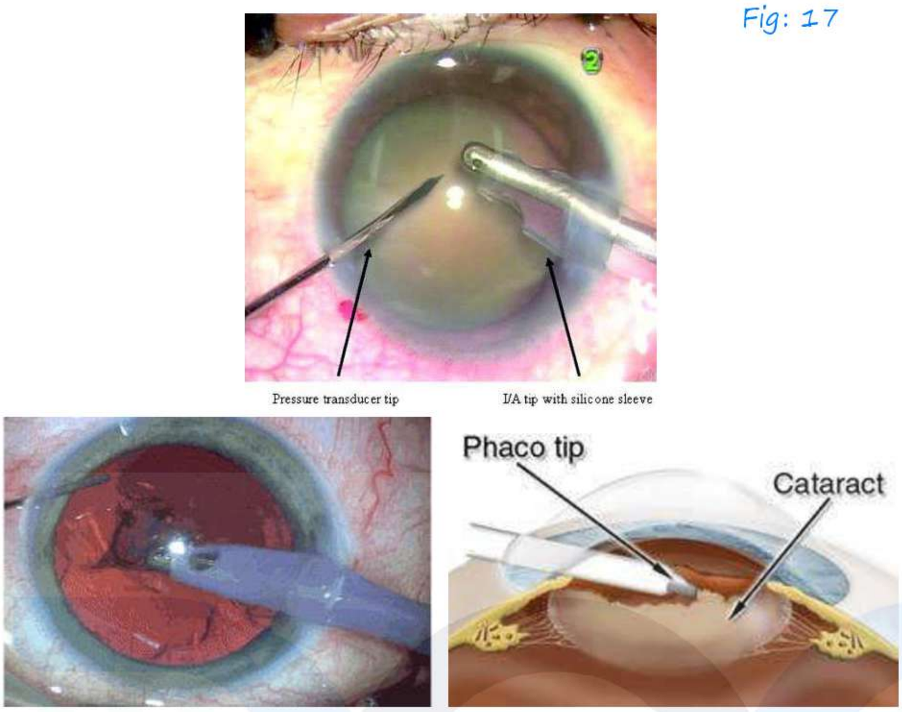
Fig: 16

↓

Hydro delineation → separates nucleus from cortex

- Golden ring – sign of complete delineation

Fig: 17


Pressure transducer tip
I/A tip with silicone sleeve
Phaco tip
Cataract

- Phacoprobe has titanium needle which has piezoelectric crystals ultrasonic power
  - Vary at frequency of 4000HZ

Fig: 18

- Inject the lens
- Closure of ports

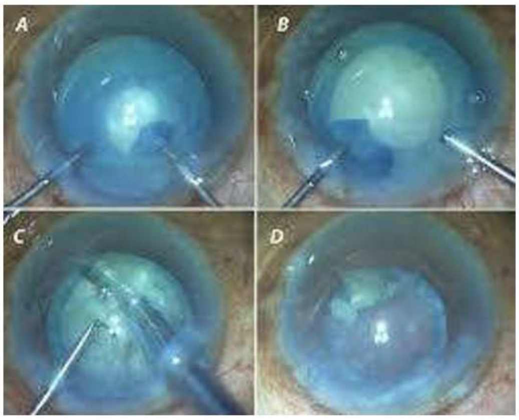


Fig: 19

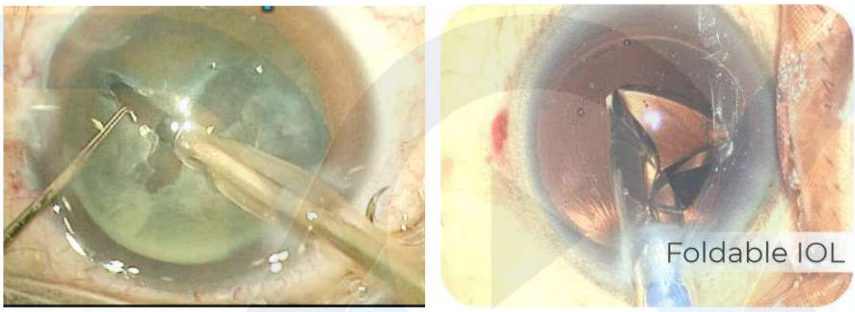


Fig: 20

Advantage of phacoemulsification

- Smaller incision
- Less astigmatism

Modification

- MICS

↓

Micro incisional cataract surgery

↓

Size < 2 mm

FLACS – Femtolaser assisted cataract surgery

00:34:00

- The femtosecond – ultrafast pulses in the range Of 10^{-15}
  - Highly coherent beam of laser is focused at 3mm spot size with accuracy of within 5µm
  - Photo-disruption is induced by a state of plasma that is generated by the vaporization of target tissues.
  - The force of cavitation bubble creates separation of tissue.

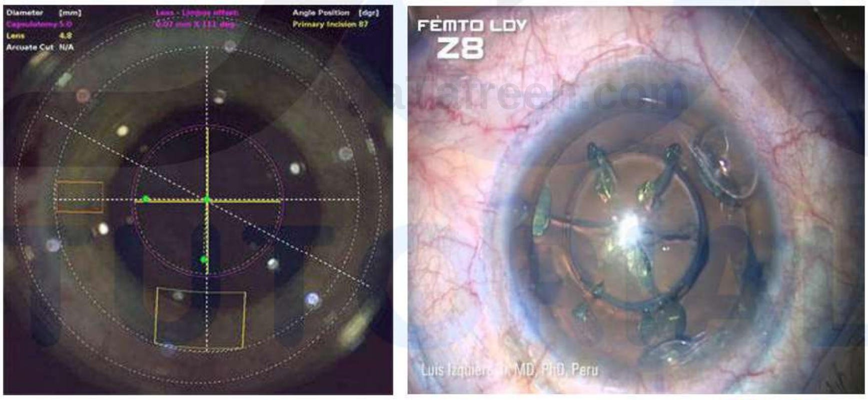

Diameter [mm]
Capsulotomy 5.0
Lens 4.8
Arcuate Cut N/A
Angle Position [dgr]
Primary Incision 87
FEMTO LDV
Z8
Luis Izquierdo Jr, MD, PhD, Peru

Fig: 21

- FLACS – Femtolaser assisted cataract surgery

  ↓

  Utilizes Nd glass – wave length of 1053 nm

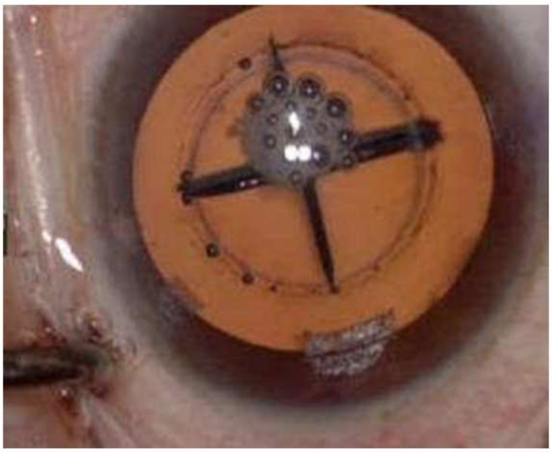


Fig: 22

Steps by Femto Laser

- Used to create main port
- Used to create side port
- Used to create CCC (perfect centered)
- To create nucleotomy

TYPES OF INTRAOCULAR LENS

- Sir Nicholas Harold Lloyd Ridley was an English ophthalmologist who invented the IOL
- November 29, 1949 at ST. Thomas Hospital, London
- 49 years woman

Fig: 1

Types of Intraocular Lens 00:01:05

i. On Basis of site of placement 00:01:15

- Posterior chamber within capsular bag (ideal)

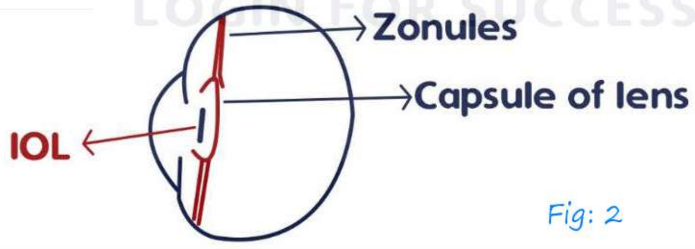


Fig: 2

- Sulcus – space between iris and anterior capsule
- Anterior chamber IOL

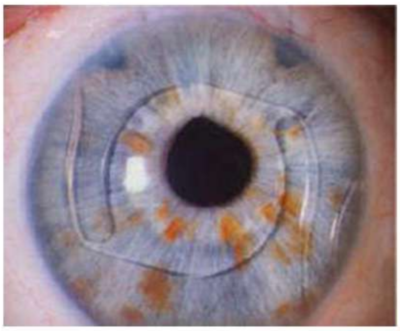
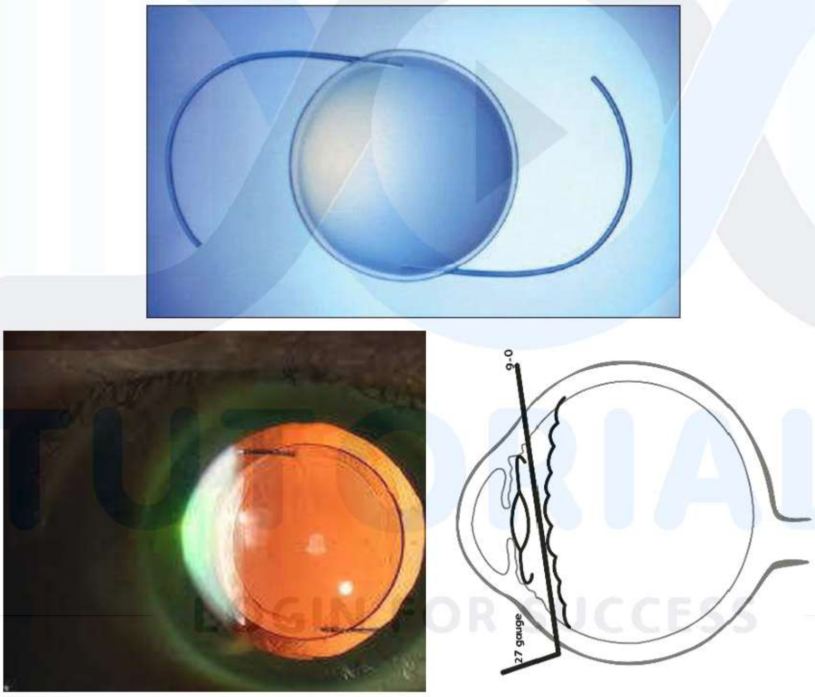


Fig: 3

- Causes U- Uveitis
  G- Glaucoma
  H- Hyphemia



Posterior chamber IOL

Fig: 4

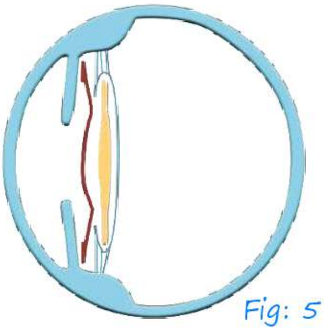


Fig: 5

- If none of them available → SF IOL → scleral fixated intraocular lens

Advantage of in bag placement of IOL 00:05:50

- Proper anatomical site
- Intraoperative stretching or tearing of zonules is avoided
- Minimal magnification
- Low incidence of lens decentration and dislocation
- Reduced posterior capsular opacification

ii.On Basis of material 00:06:50

- Rigid lens- made of PMMA – polymethyl methacrylate
- Foldable ⟶ Acrylic → Best and most common used
  ↘ Silicone→ discouraged in patients were high myopia,
  diabetic retinopathy

TUTORIALS
LOOK FOR SUCCESS

Hydrophobic vs Hydrophilic

Hydrophobic	Hydrophilic
Hydrophobic lenses have a low water content (<1%)	Higher tissue compatibility due to higher water content
High flare	Low flare/reaction
Less chances of PCO	More chances of PCO

Hydrophobic lens

- Most common used

Rollable IOLs

- ultrathin~ 100μ
- hydrophilic material
- implanted by phakonit technique

iii. On Basis of focal length 00:09:35

- **Monofocal** ~ most commonly used lens, corrects only distant vision
- Accommodative function is lost

↓

Wear glasses to read

Multifocal lenses

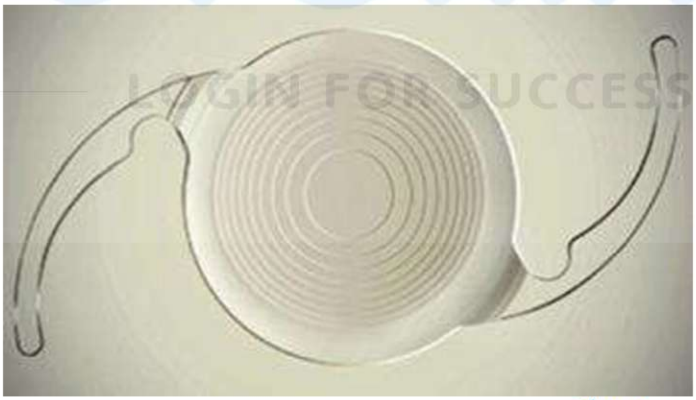


Fig: 6

↓

- Has different segments

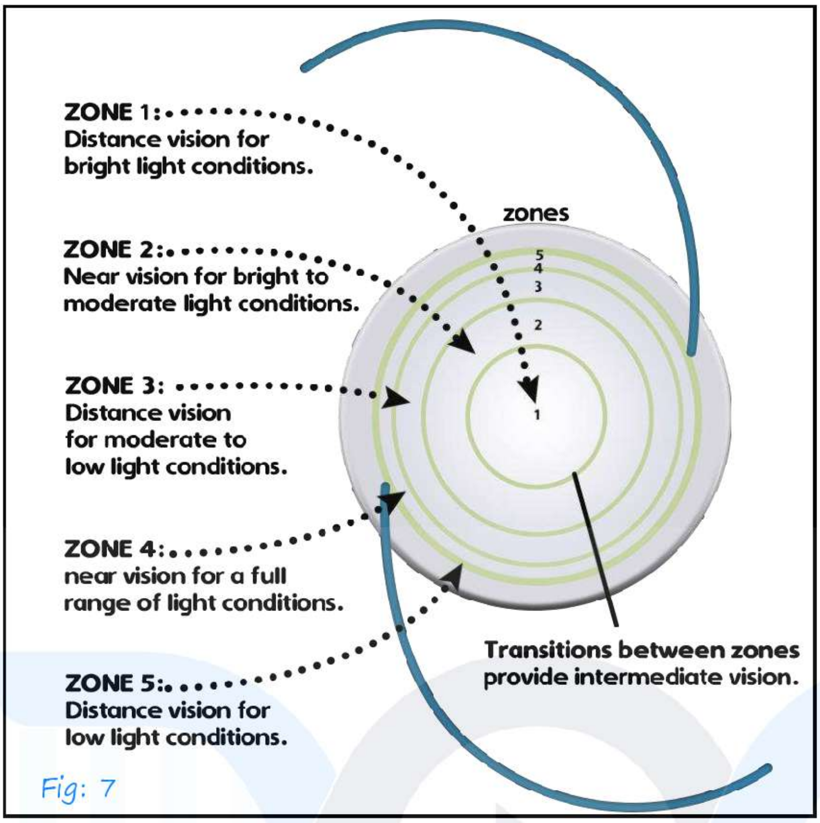

ZONE 1:
Distance vision for bright light conditions.
ZONE 2:
Near vision for bright to moderate light conditions.
ZONE 3:
Distance vision for moderate to low light conditions.
ZONE 4:
near vision for a full range of light conditions.
ZONE 5:
Distance vision for low light conditions.
zones
5
4
3
2
1
Transitions between zones provide intermediate vision.

Fig: 7

Fig: 8

Atfal afreeh.com
DOC TUTORIALS
FOR SUCCESS

Refractive multifocal IOLs

Bull's eye lens

- Concentric rings of different power

Diffractive multifocal IOLs

- Anterior aspheric surface basic refractive power multiple on posterior surface
- Refractive lenses (pupil dependent)
  - Drive during the day

  - play sports
  - Use a computer frequently or activities
  - That rely heavily on intermediated vison
- Diffractive IOLS (pupil independent) for
  - Spend a lot of times reading
  - Detailed craft work
  - Scotopic activities
  - Movies
  - Night time driving
- problems → glare/halos

Special type of IOLs 00:15:20

Fig: 9

TUTORIALS LOGIN FOR SUCCESS

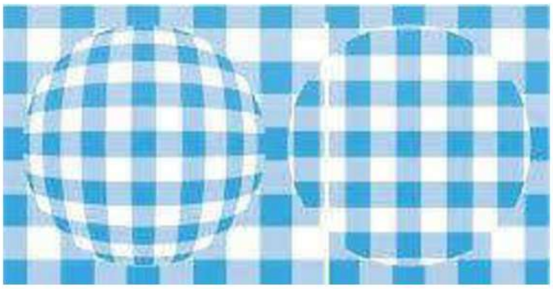


Fig: 10

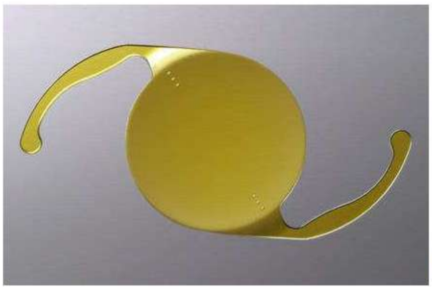


Fig: 11

- Square edge

  ↓

  Prevents PCO formation
- Aspheric → corrects spherical aberration
- Heparin coated → decreases the chance of uveitis
- Toric IOL → Correct the preexisting astigmatism

  → It aligns with most spherical region
- Blue blocking → useful for ARMD patients

COMPLICATIONS OF CATARACT SURGERY

Per operative	Early post operative	Late post operative
Vitreous loss	UGH Syndrome	Posterior capsular opacification
Posterior capsular rupture	Endophthalmitis	Cystoid macular edema (Irvin Gass Syndrome)
Nucleus drop	Tass	Retinal Detachment
IOL drop	Shallow anterior chamber wound leak	Late endophthalmitis
Suprachroidal hemorrhage	Malignant glaucoma	IOL related complications
Descemet membrane detachment	Corneal edema	Fibrous downgrowth

- During surgery, IOP may become very high
  Bleeding in supra choroidal space
  ↓
  Supra choroidal hemorrhage
- Vitreous may come out through defect or posterior capsule or zonules
  ↓
  Vitreous loss
- Torture to Descemet's membrane
  ↓
  Descemet membrane detachment
- Early postoperative complications → < 3months
- Late postoperative complications → > 3 months

UGH syndrome

Uveitis ┐
Glaucoma ├ syndrome
Hyphema ┘

- During cataract surgery, manipulation is there,
  - Inflammation
  - Increase IOP
- UGH syndrome present in any cataract surgery, more with ACIS

Endophthalmitis & TASS

↓

- Can happen due to severe reaction in anterior chamber and posterior segment involved
- Most common complication generally → Posterior capsular opacification
- Cystoid macular edema can occur as a part of inflammation to retina
- Irvin gars syndrome → seen after ICLE + vitreous loss

Endophthalmitis 00:04:19

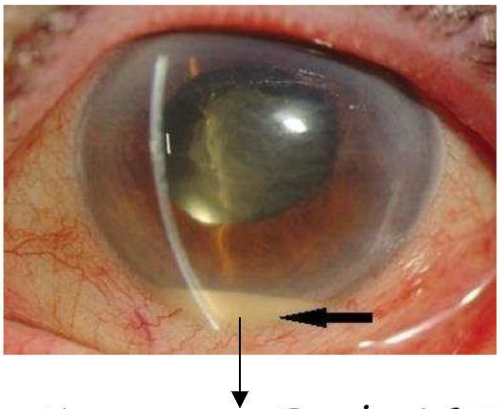
- Endophthalmitis → Severe inflammation of inner contents of eye with vitreous exudation
- Sclera is spared
- Can be due to ┬ exogenous (most common)
  └ endogenous (rare)

- Exogenous → trauma and postoperative (most common)
- Postoperative endophthalmitis

Early
↓
- Staph. epidermidis (CoNS) coagulase negative staph.

Late
↓
- Fungus
- Propionibacterium acne

- Symptom
  - Painful decreased vision
  - Redness
  - Photophobia
- Signs
  - Cornea is hazy
  - Anterior chamber reactions
  - Hypopyon
  - Vitritis
- USG – B done to look for vitreous exudation
- It is an ocular emergency



Hyoopym – Pus in AC Fig: 1

- EVS → Endophthalmitis vitrectomy study
  - Said that if the vision is poor, but better than PL ⊕ PR acc

    ↓

    Intravitreal antibiotic → TOC
  - If vision is poor, less than PR (inaccurate)

    ↓

    Vitrectomy (pars plana vitrectomy)
- No role of systemic antibiotics
- Cycloplegics ⊕

Toxic Anterior Segment Syndrome (TASS)

00:12:57

- Generally, occurs on 1st post operative day
- Inflammation is only in anterior segment
- Corneal edema → more diffuse, White-white limbal area
- Responds very well to topical steroids

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LOGIN FOR SUCCESS

Late endophthalmitis 00:15:41

- P. acne causes inflammation late after cataract surgery, but the typical of this is produces a plaque on IOL
- Treatment: Pars plana vitrectomy + removal of IOL

Posterior capsular opacification 00:16:46

- Most common complication of cataract surgery
- Late complication

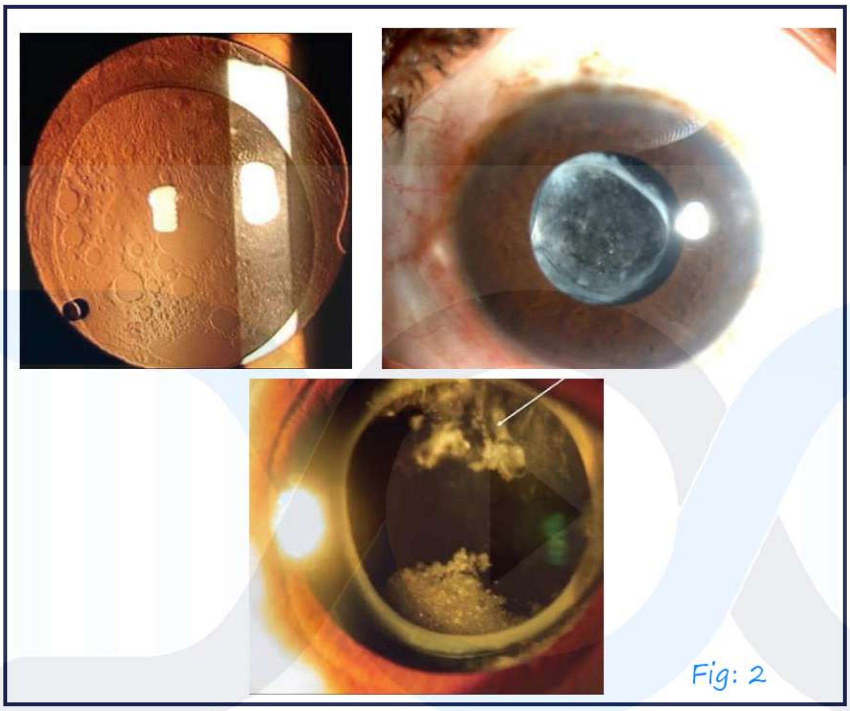
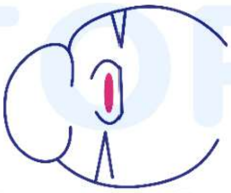


Fig: 2



Posterior capsule develops few opacification areas

- 2 types
  - Elschnig pearls → vacuolated bladder most common
    - wedl cells
  - Sommerring ring
- After cataract / 2° cataract as symptoms are same as that of cataract

Treatment

- small hole made in posterior capsule

  ↓

  Nd YAG posterior capsulotomy

  ↓

  Wavelength of 1064nm

  Mechanism of photo description

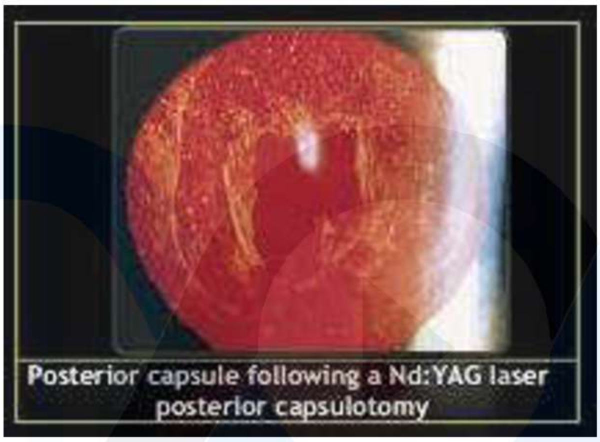

Posterior capsule following a Nd:YAG laser posterior capsulotomy

Fig: 3

IOL related complications 00:22:32

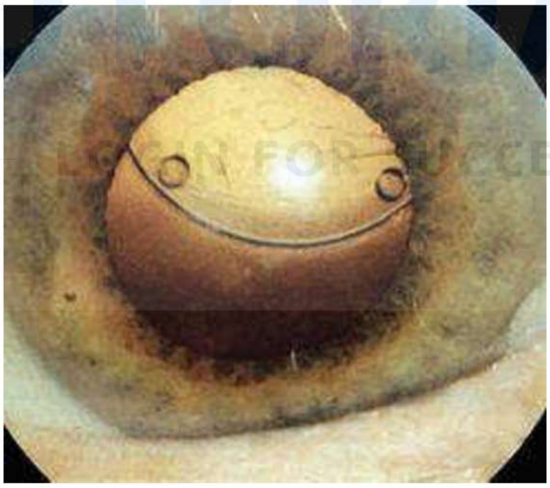


Fig: 4

- sunset syndrome – inferior subluxation of IOL
- sunrise syndrome – superior subluxation of IOL

- lost lens syndrome → Zonules are weak, lens may be completely dislocated
- wind shield wiper syndrome

  ↓

  when the IOL is too small for the eye and the superior haptic moues with head movements.

- If lens has gone up and out → inherent weakness of zonules is there
- Upper zonules are strong and pull up causes of subluxation

Subluxation (BPL ME H S H) Below Poverty Line ME Hum Subluxate Hue

- B – Buphthalmos
- P – PXF
- L – Hyperlysenemia
- M – Marfans, Marchesani syndrome
- E – Ehler Danlos Syndrome
- H – Homocystinuria
- S – Sulphite oxidase deficiency
- H - HMSC

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Marfan syndrome

- Supertemporal subluxation → Most common ocular feature
- Other features:
  - Megalocornea
  - cornea plana
  - cataracts
  - squints
  - strabismus
  - Refractive error

Homocystinuria → Inferonasal subluxation

DISLOCATION OF LENS, CONGENITAL CATARACT

Subluxation and dislocation of Lens 00:00:12

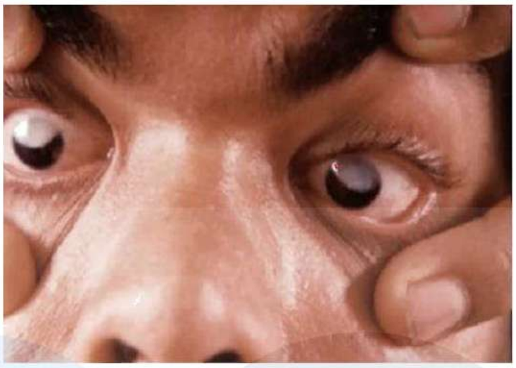


Fig: 1

- Lens going up and out → Subluxation → Displacement of lens
- Dislocation → Complete displacement of lens from its normal place (Patellar fossa)
- C/O diplopia → due to Aphakic area & phakic area
- Edge of lens never seen

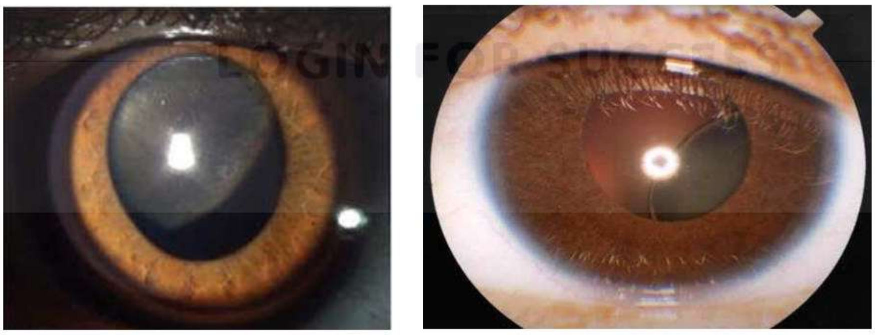


Fig: 2

- DDO – Distant direct ophthalmoscopy
  - If edge of lens seen → there is subluxation
- There are Several causes for dislocation of lens (+)
- Baric cause – zonular support is not proper

Weil marchesani syndrome

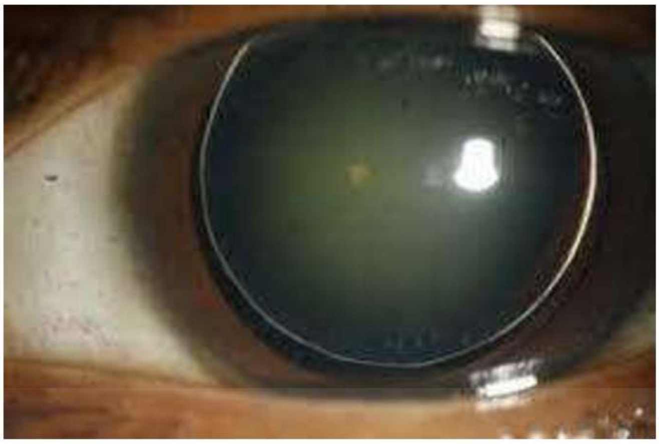


Fig: 3

- Short stature, progressive joint sriffness, brachydactyly, thick skin, mild mental retardation, and cardiac anomalies.

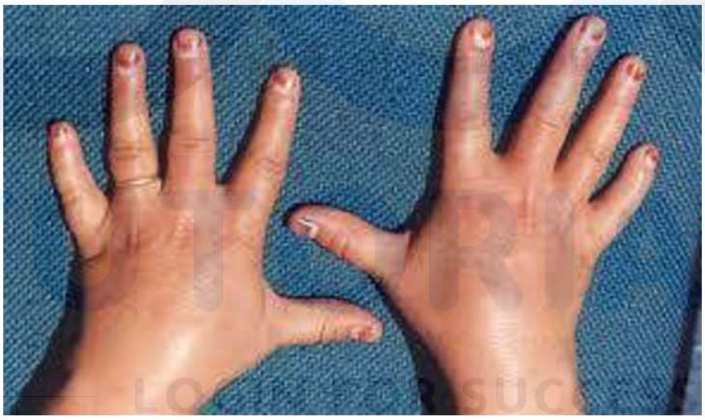


Fig: 4

- Pupillary block is exacerbated with miotics and relieved by mydriatics.
- Miotics cause ciliary muscle contraction and loosening zonular support, allowing forward movement of the lens and shallowing of the anterior chamber, eventually leading to pupillary block.
- Short stature

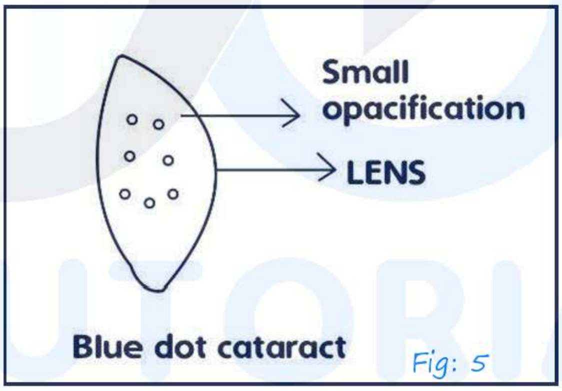
TUTORIALS LOGIN FOR SUCCESS

- Microspherophkia → Zonules are long and slightly laxed
- Complication → Inverse glaucoma → aggravated by miotics → Pilocarpine
  - Improves with cycloplegics

Congenital Cataract 00:09:10

Infantile → Congenital/At birth/<1yr of age
Developmental → after 1 year of birth

- Most common congenital cataract → Blue dot cataract



Blue dot cataract Fig: 5

- Vision not impaired
- Universal cataract
- Also called as considerable cataract

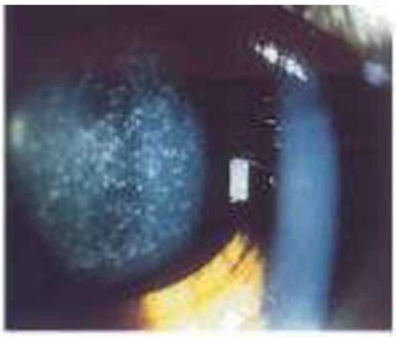
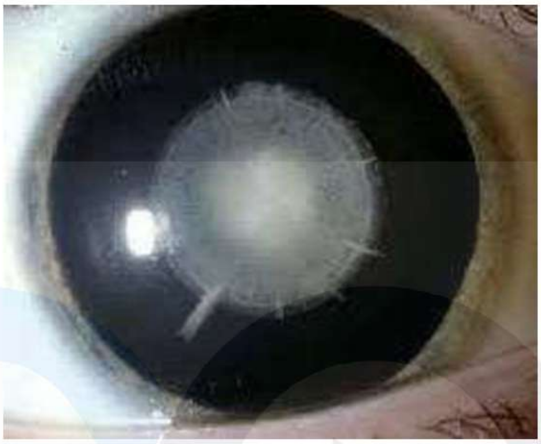


Fig: 6

blue dot / cataracta coerulea

- Most common congenital cataract associated with decrease in vision → zonular cataract/Lamellar cataract
- Lamellar cataract → Riders (+)



Lamellar/Zonular Fig: 7

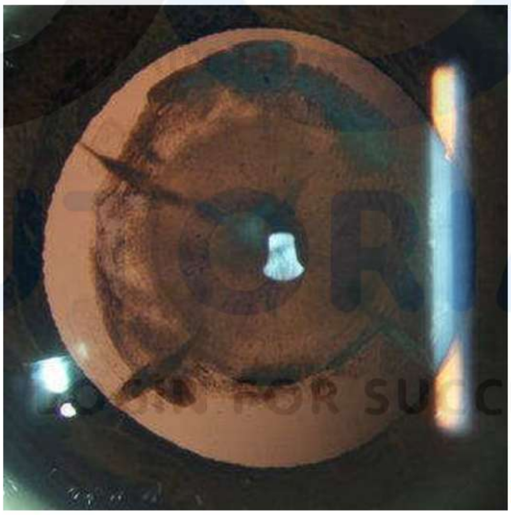


Fig: 8

Zonular cataract with riders/

Cart wheel = spoke of wheel

- It basically depends on which part of nucleus is involved

TUTORIALS FOCUS FOR SUCCESS

- Pregnant female and she captures rubella 5th month of pregnancy → Child can have congenital cataract
- 0-3month – embryonic zone
- Insult has occurred in 5th month – Fetal zone involved → Opacity in fetal zone

- Zonular cataract also associated with
  - Maternal rubella
  - Vit D deficiency
  - Zonular – Riders giving spoke of wheel appearance

Sutural

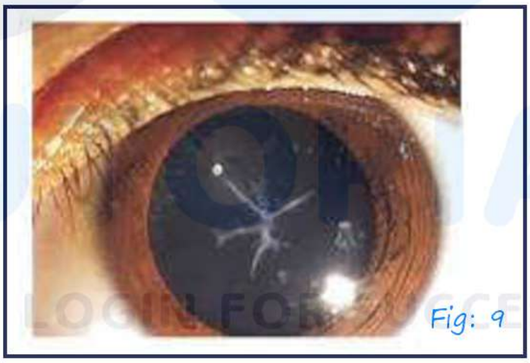


Fig: 9

- Opacity located within the sutures (Y shaped sutures)

Cataract centralis pulverulenta

- Type of punctate cataract
- Small white dots

Nuclear - embryonic rubella

- Pearly nuclear cataract
- Congenital rubella → Ocular features present if infection is in rubella patients
- Virus persists in lens up to 3years
- The child can have congenital glaucoma, retinopathy, microphthalmic eyes

Coronary

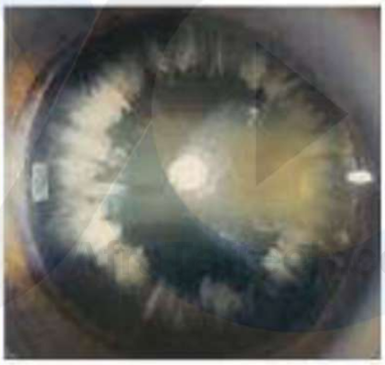


Fig: 10

- Club shaped capacities in periphery

Anterior polar cataract

- Usually after a perforated corneal ulcer

Posterior polar cataract

- Onion peal appearance

Treatment

- Unilateral cataract → C/O leukocoria

  ↓

  dense – interferes with vision
  Squint (+)
  ] Surgery (As soon as possible)
  ↓
  To prevent amblyopia

- Bilateral cataract

  ↓

  May not be dense
  Decreased vision
  Squint (+)
  pupillary axis
  ] Surgery (As soon as possible)

- Surgery → lens aspiration + Posterior continuous curvilinear capsulorhexis + Anterior vitrectomy

  ↓

  Prevent further PCO formation → Posterior capsular opacification

  IOL placed for > 6months of age

- In <2years of age → Under correct by 20%

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Axial length	IOL power
17mm	25D
18mm	24D
19mm	23D
20mm	21D
21mm	19D

- In 2-8 years → under correct by 10%

SCLERA

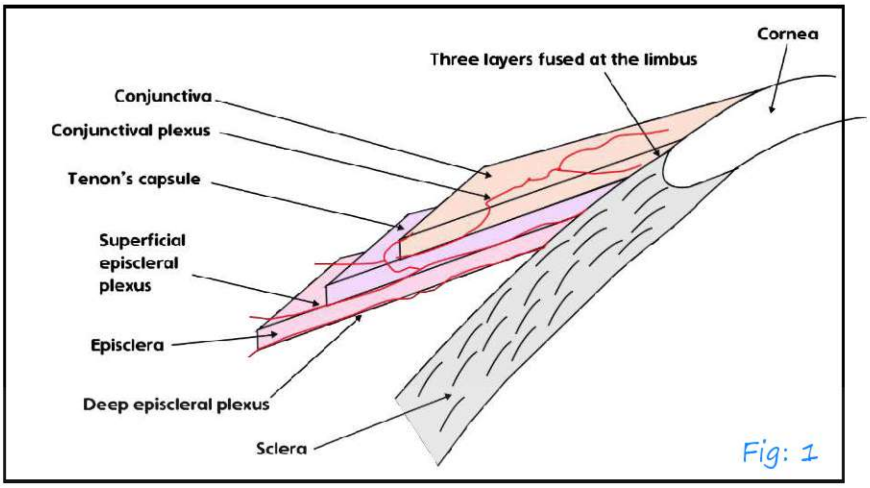

Cornea
Three layers fused at the limbus
Conjunctiva
Conjunctival plexus
Tenon's capsule
Superficial episcleral plexus
Episclera
Deep episcleral plexus
Sclera

Fig: 1

- Sclera – White portion of the ye
  - Outer fibrous layer
- 3 layers are fused at limbus → Conjunctiva
  → Tenons capsule
  → Episclera

DOC TUTORIALS
LOGIN FOR SUCCESS

Layer of Sclera 00:01:41

- Episclera
- Sclera proper
- Lamina fusca

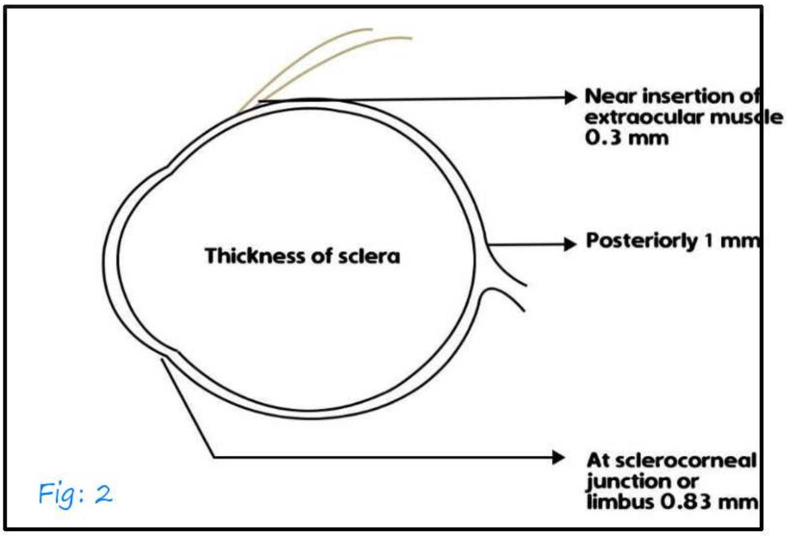

Thickness of sclera
Near insertion of extraocular muscle 0.3 mm
Posteriorly 1 mm
At sclerocorneal junction or limbus 0.83 mm

Fig: 2

- Sclera → Thickest posteriorly around the edge of optic nerve

  ↓

  1mm

  → Thinnest at just posterior to insertion of recti

  → 0.3 mm

Episcleritis Vs Scleritis 00:03:27

Episcleritis	Scleritis
Inflammation of episcleral vessels	Inflammation of deep scleral vessel
Young adults mostly	Older individuals
Painless usually	Painful, radiation to jaw, photophobia+
Localized, diffuse, nodular	Necrotizing or non-necrotizing
Self-limiting, topical NSAIDS	Treatment required topical and oral steroids
Generally, not associated with systemic disease Gout, psoriasis, roseate, hypersensitivity to Tuberculin protein	Systemic association seen Autoimmune, RA, Wegener, PAN, AS, SLE, GOUT, HZO, Hansen's VKH, sarcoidosis
Pretty common	Uncommon

TUTORIALS LOGIN FOR SUCCESS

- Episcleritis → inflammation of superficial & spaces episcleral vessels

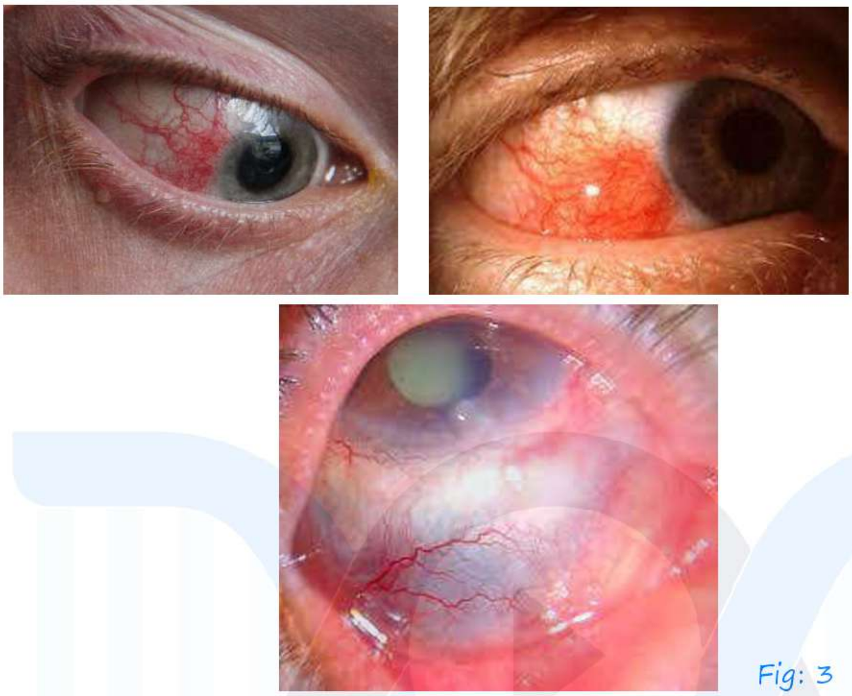
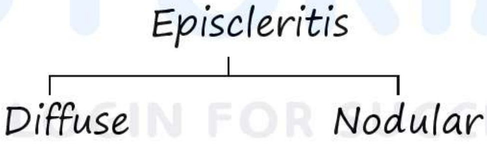


Fig: 3

→ Diffuse

→ Nodular


Episcleritis
Diffuse
Nodular

BEGIN FOR SUCCESS

Scleritis

Anterior

Posterior

Necrotizing with inflammation

Necrotizing Co/ O inflammation

↓

Scleromalacia perforance

↓

Usually seen in elderly female

- May be decrease in vision
- Not painful → inflammation because of ischemic necrosis of arteries

↓

Sequences formation

↓

White: painless

- Sclera generally appears white /thinned /blue

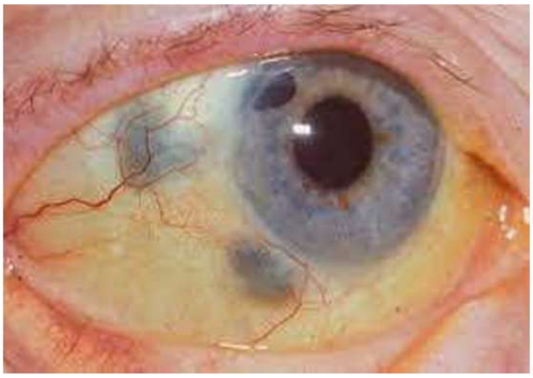


Fig: 4

- RA long standing
- No redness
- White
- Bluish discoloration of sclera → Sclera becomes thin and choroids are visible

Posterior scleritis

- Posterior to equator
- Pain
- Globe tenderness
- Decrease in vision
- Proptosis
- Exudative retinal detachment
- Retinal → choroidal folds seen
- USG B scan: – 'T' sign

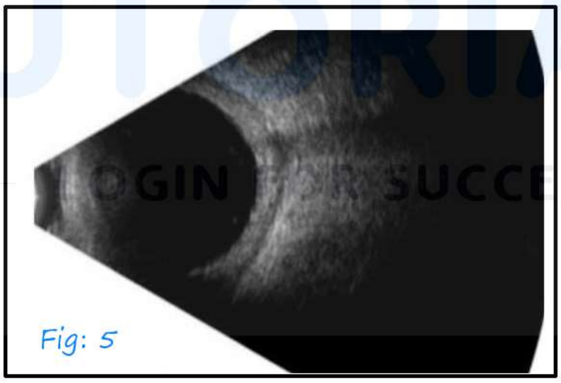
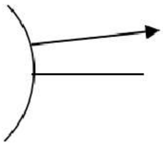


Fig: 5



→ due to fluid collection in subtenon space

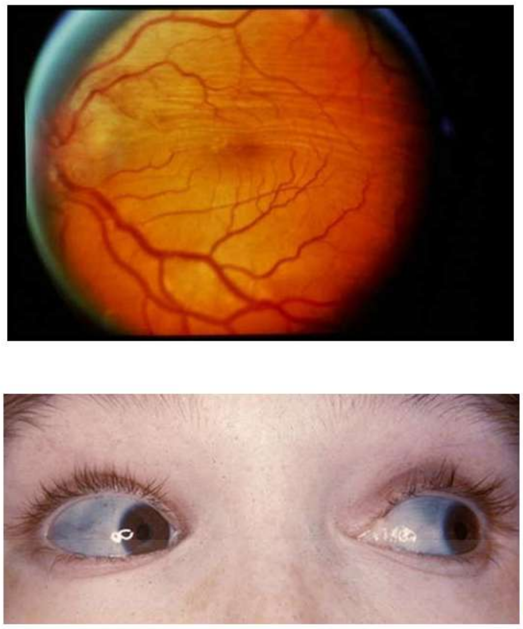


Fig: 6

Causes of blue sclera: BOSS HAVE M2 ulti P2 olar Disorder

- Buphthalmos, Osteogenesis imperfecta, scleritis, staphyloma'
- Hypophosphatasia, Alkaptonuria, Van der hoeve's syndrome, Ehlers Danlos syndrome
- Myopia, Marfan's, Paget's disease, Pseudoxanthoma elasticum, diamond backfan syndrome

Staphyloma 00:16:32

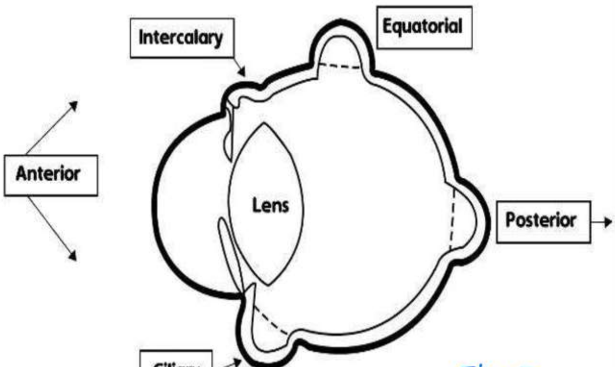

Intercalary
Equatorial
Anterior
Lens
Posterior
Ciliary

Fig: 7

Definition

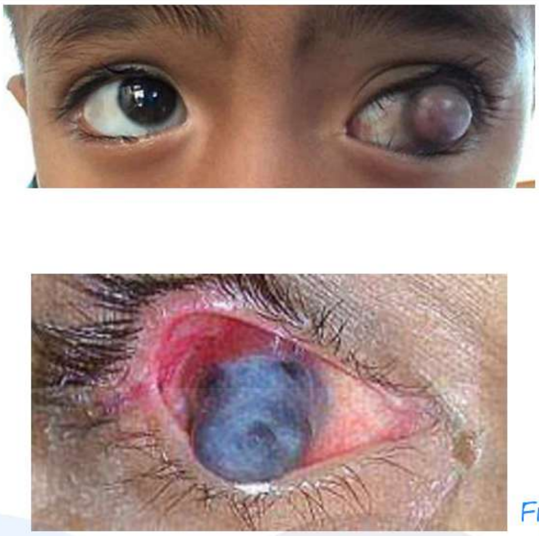
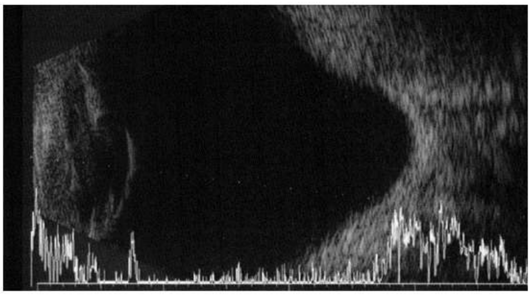


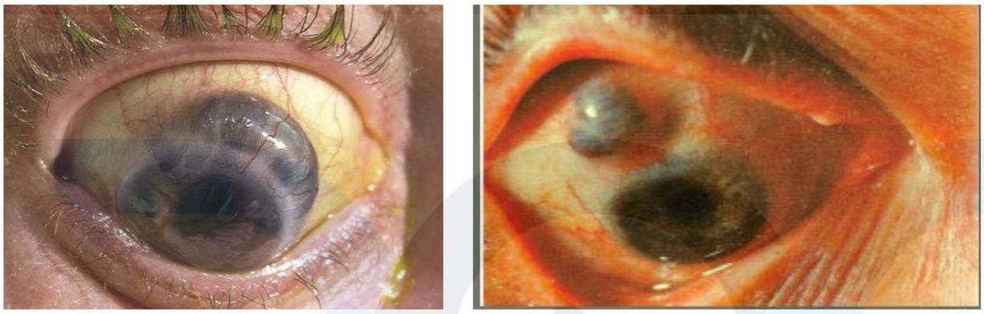
Fig: 8

- Staphyloma is an ectasia of the outer coat eyeball with underlying uveal tissue
- 5 type anterior → cornea + iris

Staphyloma	Location	Comments
Anterior	Over cornea, underlying iris	MC cause; perforated corneal ulcer
Intercalary	Within 3 mm to limbus, underlying root of iris	MC cause: peripheral ulcerative Keratitis, traumatic perforation
Ciliary	After 3 mm from limbus, underlying ciliary body	MC cause: scleritis, perforation, absolute glaucoma
Equatorial	At equator of eyeball, underlying choroid	MC cause: Scleritis, Myopia
Posterior	Behind equator, posterior pole	MC cause: Pathologic Myopia



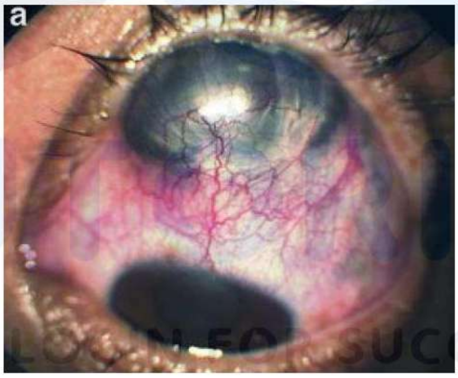
USG B scan O cupping Fig: 9



Intercalary

Ciliary

Fig: 10



Equatorial Fig: 11

INTRODUCTION – ANATOMY LAYERS

- Reddish orange in appearance (due to vascular choroid behind retina transparent in color
- Outer layer of retina (Retinal pigmental epithelium) is different from the inner 9 layers (neurosensory retina), developmentally
  - There is a gap between NSR and RPE< caused a subretinal space
  - Neuroectodermal in origin
  - Differential growth of the neuroectodermal cup
- The gap between RPE and choroid is caused a sub RPE space

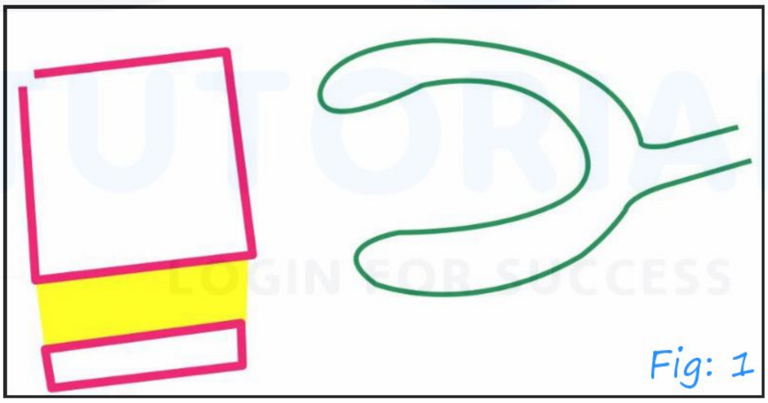


Fig: 1

TUTORIALS LOGIN FOR SUCCESS

Layer of Retina (10 layers)

i. Retinal pigment epithelium (outermost layer)
   a. Single layer of cells
   b. Melanin-present; absorbs harmful UV rays and nerves back scattering of light
   c. Forms outer blood – retinal branle

  d. Phagocytic function

ii. Layer of photoreceptors out segment of
  a. Increase Rods and cones
  b. Rods – 120 million cones- 6 million
  c. Generation of local graded potential
  d. Rods and cones are the first order neurons for the visual pathway

iii. External limiting membrane
  a. Not a true membrane
  b. Limits outer segment of photoreceptors from nuclear
  c. Made of cell processes of muller cells and photoreceptor cells

iv. Outer nuclear layer
  a. Nuclear of rods and cones

v. Outer plexiform layer
  a. Synapse of axons of photoreceptors and dendrites of bipolar cells, muller cells, amacrine cells, horizontal cells (mainly)
  b. Watershed area (receives blood supply from both the chordal side and central retinal vessels)
  c. Collections of fluids /exudates – happens in this layer generally
  d. The cells of OPL have a different configuration at the macula → the layer

vi. Inner nuclear layer

a. Nuclei of bipolar; muller cells, amacrine cells, horizontal cells

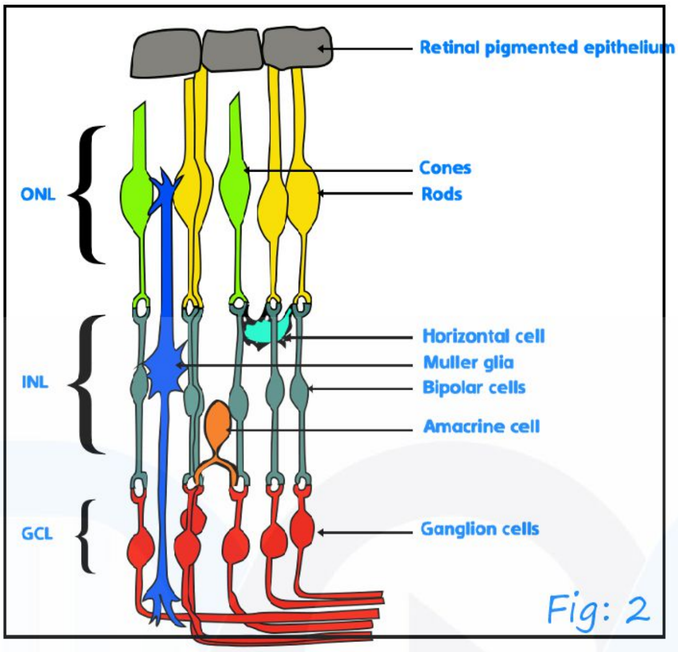

Retinal pigmented epithelium
Cones
Rods
ONL
Horizontal cell
Muller glia
Bipolar cells
INL
Amacrine cell
GCL
Ganglion cells

Fig: 2

b. Higher tendency for microaneurysm

c. Bipolar cells→ 2nd order neurons for the visual pathway

d. Muller cells→ supporting cells, present in almost all layer of retinal except RPE and photoreceptors
   - Phagocytic action
   - Metabolism

vii. Inner plexiform layer

   a. Synopsis of axons from bipolar cells and dendrites

   b. From a ganglion cell

viii. Ganglion cell layer

   a. Nuclei of ganglion cells (third order neuron for visual pathway)

ix. Nerve fiber layer

AlfaTaleem.com
TUTORIALS
LOGIN FOR SUCCESS

  a. Axons from ganglions cells and from optic nerve bunter

x. Inner limiting membrane

  a. Limits retina from vitreous chamber
  b. True pas +ve membrane
  c. Formed mainly by foot processes of nuclear cells

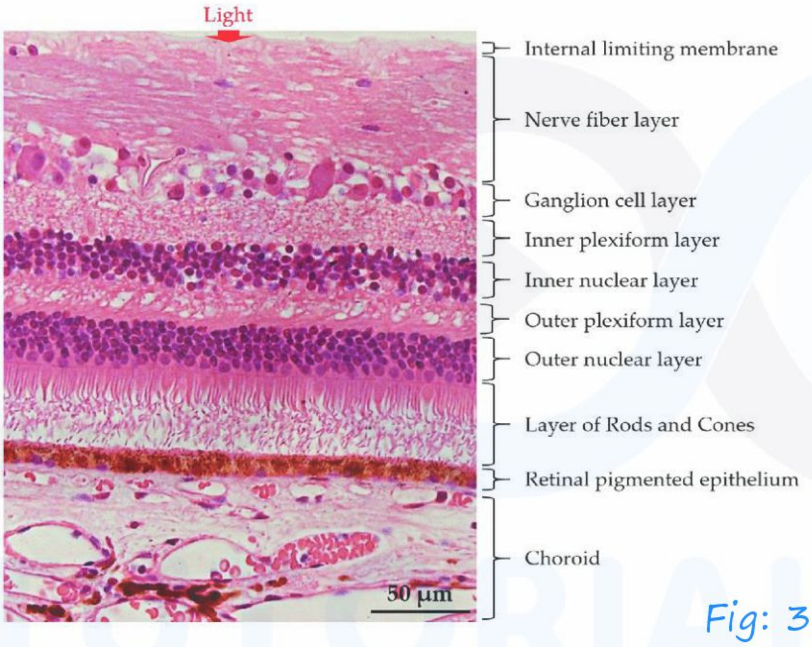

Light
Internal limiting membrane
Nerve fiber layer
Ganglion cell layer
Inner plexiform layer
Inner nuclear layer
Outer plexiform layer
Outer nuclear layer
Layer of Rods and Cones
Retinal pigmented epithelium
Choroid
50 µm

Fig: 3

Note

- In case of RPE atrophy, die to absence of melanin the patient experience glare clinically
- Ora selecta is the anterior mot and thinnest part of retina

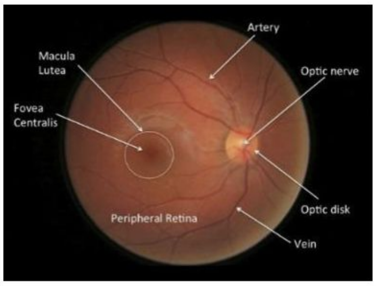

Artery
Macula Lutea
Optic nerve
Fovea Centralis
Optic disk
Peripheral Retina
Vein

Fig: 4

Anatomy of fovea

- Depressed
- Most of the layer of retina ore absent esp. Ganglion cells layer
- Clinical correlation – cherry red spot
- Fundus is visible in on ophthalmoscopy
- Anterior retina is not easily seen via an orthoscopic optic disc optic nerve head → 1 mm in width
  → 1/5 mm in diameter
- Right eye retina → draw the optic disc on the nasal side (RL) and vice versa

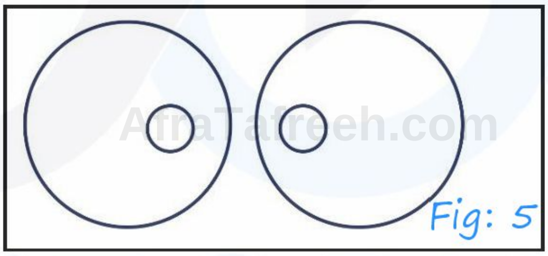


Fig: 5

- Foveal is 2-disc diameter and 0.8 mm lower temporal to the center of the optic disc → ~ 1.85/1.5 mm in diameter
- Sharpest image is formed at fovea (it has the maximum number of cones)
- 50 of the visual filed
- Zones of fovea: 1) central most 0.35 mm disc → foveola
  2) foveal avascular zone (300 to 500 μ)

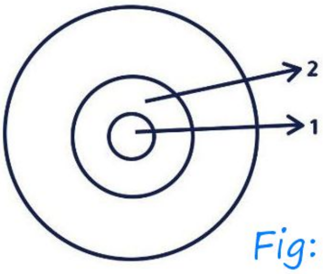


Fig: 6

- Foveal reflex→ cent almost combo (shining reflex a the canty)

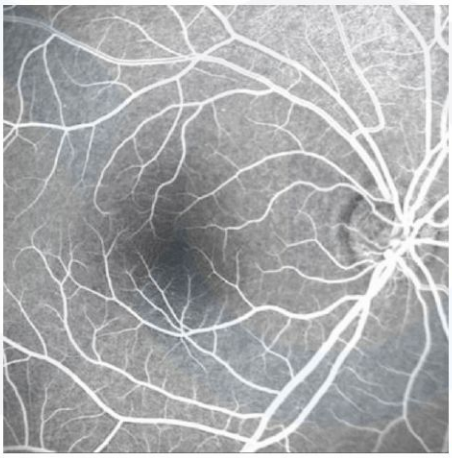


Fig: 7

- Foveal avascular zone (300-500μ)
- Xanthophyl is present → absorbs fluoresce dye
- Tall RPE cells present (which block florescent)

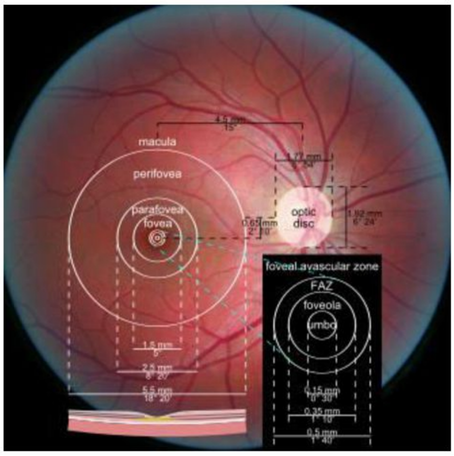

4.5 mm
15°
macula
perifovea
parafovea
fovea
1.77 mm
5° 54'
optic disc
1.92 mm
6° 24'
0.65 mm
2° 10'
1.5 mm
5°
2.5 mm
8° 20'
5.5 mm
18° 20'
foveal avascular zone
FAZ
foveola
umbo
0.15 mm
0° 30'
0.35 mm
1° 10'
0.5 mm
1° 40'

Fig: 8

- Zones outsides fovea's
  - Parafoveal zone
  - Perifoveal zone
- A circle of 5.5 mm diameter both foveal entre don it is macula ludea
- Most important region of retina responsible for central vision

Blood supply to the retina

- Supplied by central retinal artery (Brach of optimism (inner 6 layer)

  ↑

  Internal cystoid A
- Also supplied by choroidal vessels (outer 4 layers)
- Central referral artery supply

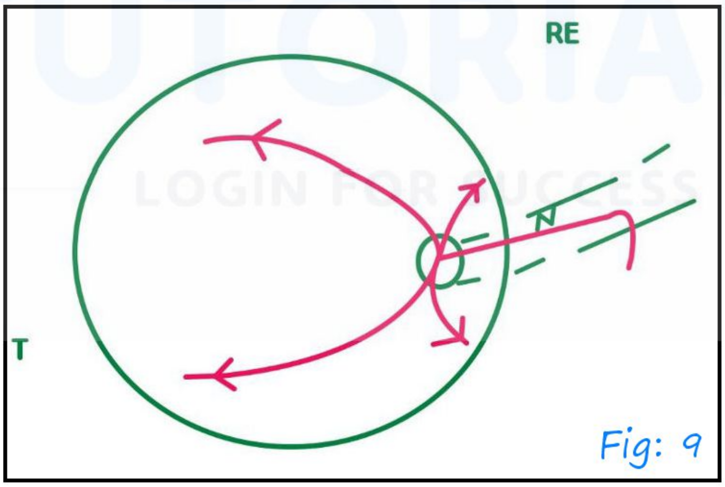


Fig: 9

- Tributaries of central retinal vein

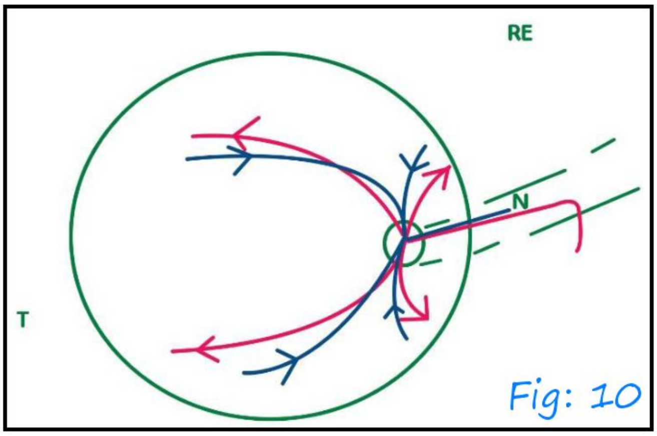


Fig: 10

Blood Retinal Barriers

Outer
↓
Tight junction of RPE
↓
Neurosensory detachment
Cantal serous retinopathy

Inner
↓
Tight junction of capillary endothelium
↓
CME
(Cystoid macular edema)s

RETINAL DETACHMENT & DEGENERATION

- It is a misnomer, there is no detachment of Retina from Vitreous/ choroid
- There is separation of NSR from RPE with collection of fluid in subretinal space

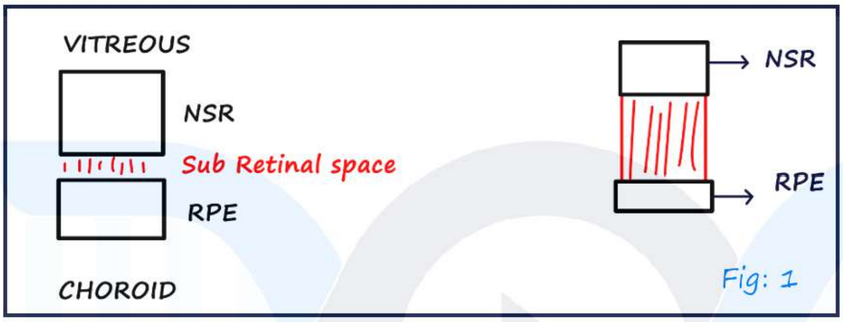


Fig: 1

Types of Retinal Detachment 00:02:35

Primary	Secondary
- Rhegmatogenous Retinal Detachment (RRDD)	- Tractional RD (TRD) - Exudative RD (Ex RD)

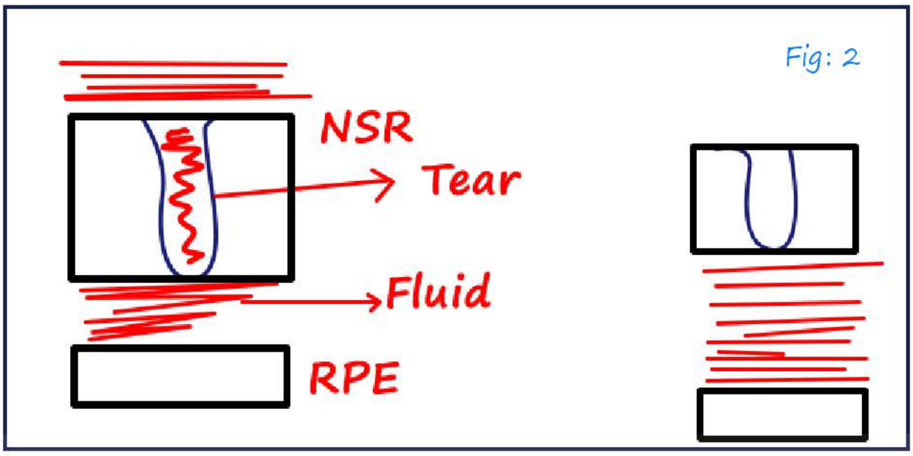
TUTORIALS LOGIN FOR SUCCESS

Rhegmatogenous RD

↓

Tear

- Most common Type of Retinal Detachment → occurs when Full thickness tear develop in NSR



Etiology

- High Myopia
- Intraocular Sx in past
- H/O Blunt trauma

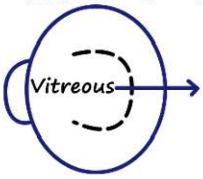
↓

Cause Early & sudden posterior vitreous Detachment

↓

- Some part of vitreous Remains attached to Retina & thus a Dynamic vitreo retinal traction is caused by it is attachment anteriorly to lens
- This is further precipitated by Head movements

Note → Normal aging



$$\text{Normally in young age “Semisolid gel”} \xrightarrow{\text{aging}} \text{Liquification}$$

+

Contraction of vitreous

↓

Detachment of vitreous from retina

Dynamic Vitreo Retinal Traction

- Can lead to full thickness Tear/Development of atrophic hole where Retina is already thinned

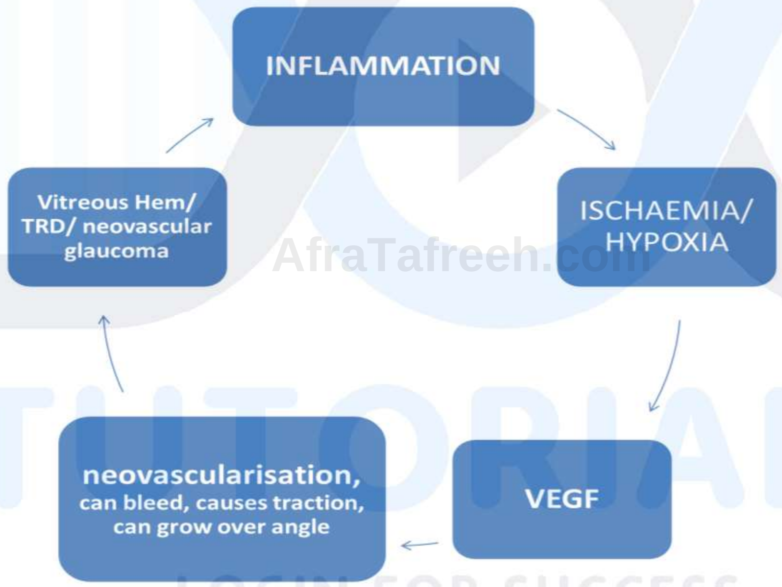
  ↓

  The liquefied vitreous will seep into the subretinal space

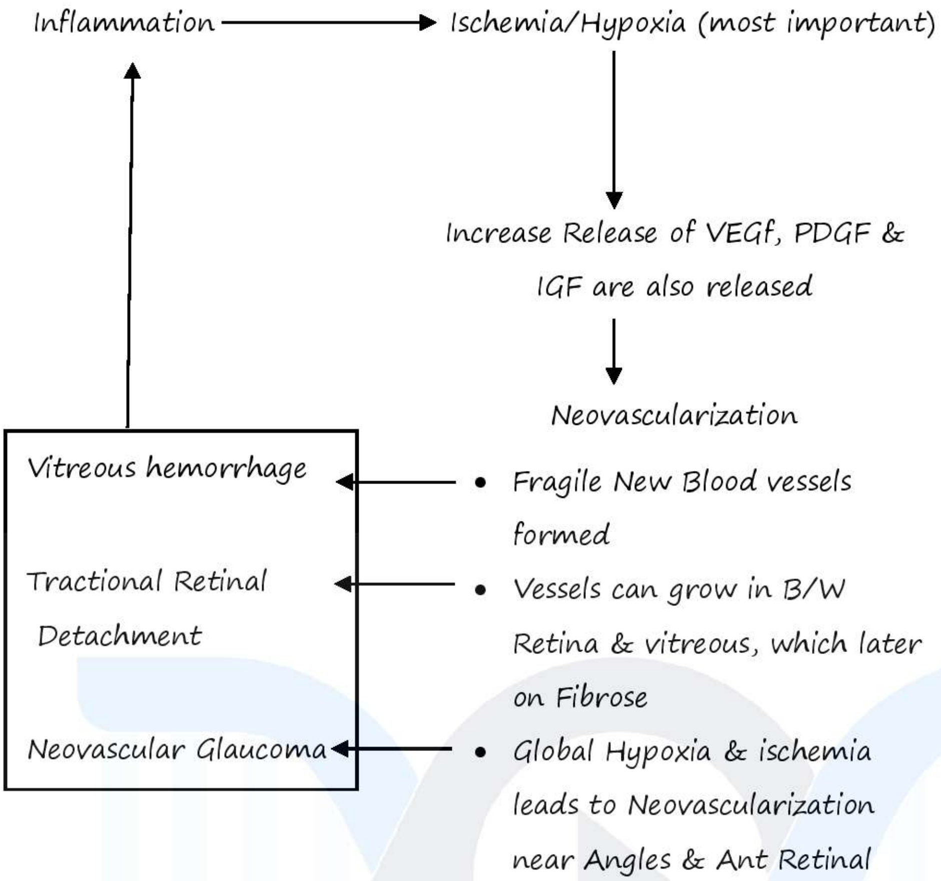
  ↓

  It leads to Rhegmatogenous Retinal detachment

Tractional Retinal Detachment:


INFLAMMATION
ISCHAEMIA/ HYPOXIA
VEGF
neovascularisation, can bleed, causes traction, can grow over angle
Vitreous Hem/ TRD/ neovascular glaucoma

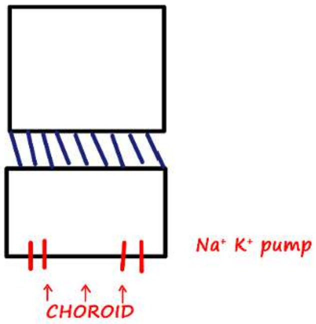
DOC TUTORIALS LOGIN FOR SUCCESS


Inflammation
Ischemia/Hypoxia (most important)
Increase Release of VEGf, PDGF & IGF are also released
Neovascularization
Vitreous hemorrhage
Tractional Retinal Detachment
Neovascular Glaucoma
Fragile New Blood vessels formed
Vessels can grow in B/W Retina & vitreous, which later on Fibrose
Global Hypoxia & ischemia leads to Neovascularization near Angles & Ant Retinal

Causes:

- Diabetic Retinopathy
- Eales Disease
- CRVO (Central Retinal venous Occlusion)
- CRAO
- Sickle cell Retinopathy
- Penetrating Trauma
- ROP (Retinopathy of Prematurity)

Exudative Retinal Detachment



↓

- Failure of Na and K PUMP) cause increase permeability & Accumulation of fluid in Sub RPE (Retinal pigment epithelium) space
- Turbid fluid is Accumulated due to choroidal Hyperpermeability/ RPE pump failure

Etiology

- Tumors of Retina /choroid
- Malignant Hypertension
- Coats disease
- Eclampsia
- Central serous Retinopathy
- Posterior scleritis
- VKH Syndrome
- Sympathetic Ophthalmia

Symptoms of Retinal Detachment:

- Sudden painless loss of vision
  (Traction Retinal detachment – gradual loss of vision)

- Field Defect: curtain over eye

Premonitory Symptoms:

i. Floaters: - Muscae volitans
  - Vitreous cells/opacities which cast image on retina & can be seen Infront of eye
  - Seen in Rhegmatogenous RD

ii. Flash: - Irritated retina send abnormal impulses to Brain which are perceived as flash
  - pt. c/o – lightening Infront of eye

    (or)

  - Thousands of Broken stars Infront of eye
  - "Floaters and flash are absent in exudative RD"

Signs of Retinal Detachment 00:22:56

i. **Distant Direct Ophthalmoscopy**:

- Normal → Red glowing

  ↓

  Grey Reflex is seen in pupillary area in RD

ii. **Schaffers Sign:**

- Seen in only in RRD

TUTORIALS LOGIN FOR SUCCESS

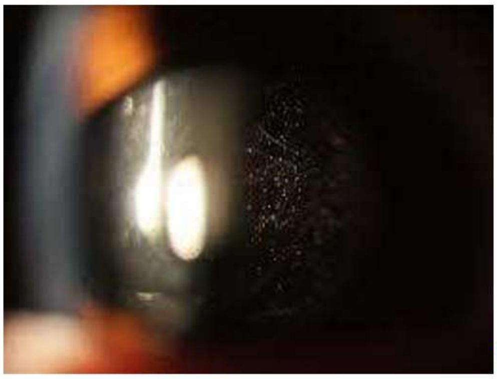


Fig: 3

- Pigment on Anterior 1/3 of vitreous

Horse Shoe shaped Tear

- Seen in indirect ophthalmoscope

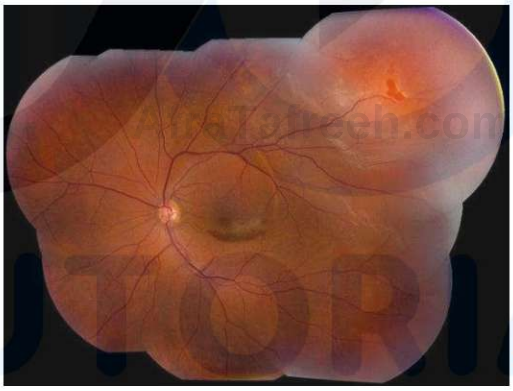


Fig: 4

Retina appearance – convex, corrugated appearance & can even extend to ora serrata in RRD

- TRD – concave, does not extend to ora serrata

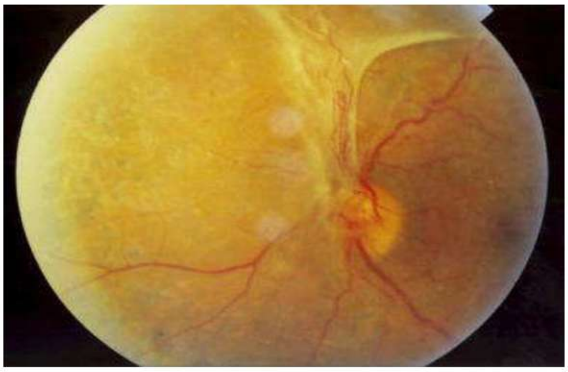


Fig: 5

- Exudative RD → shifting fluid sign present

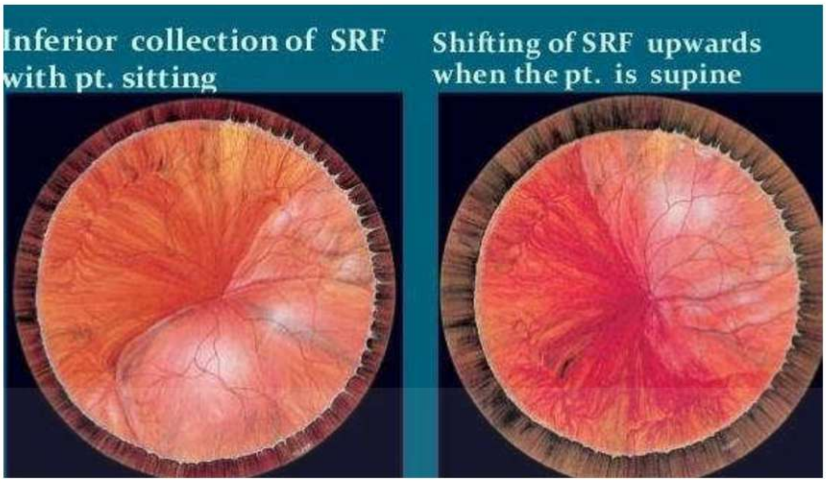
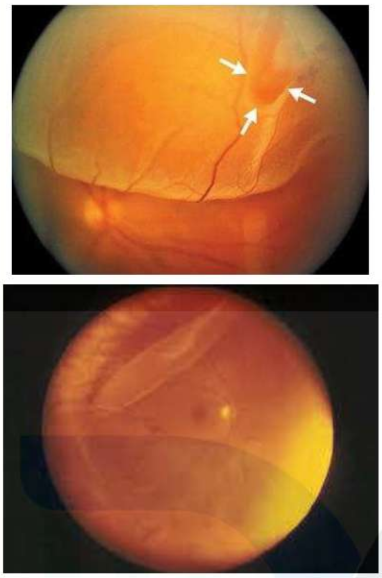
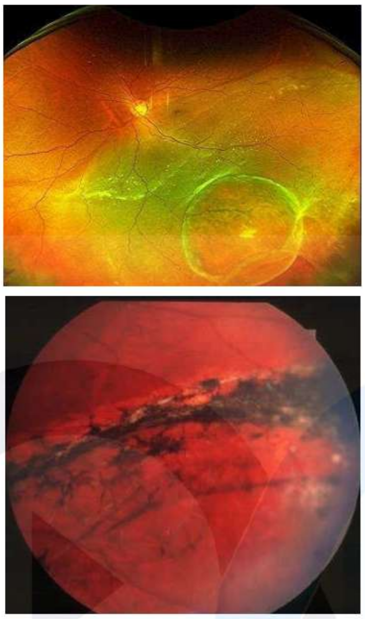

Inferior collection of SRF with pt. sitting
Shifting of SRF upwards when the pt. is supine

Fig: 6

Fresh Retinal Detachment	Old Retinal Detachment
• Convex, Deep Mobile elevation	• Retinal thinning (due to atrophy)
• Loss of choroidal pattern extending to ora serrata	• Subretinal demarcation Line/High water mark (Due to RPE proliferation) (> 3 months old RD)
• Retinal Breaks	• Secondary Intraretinal cysts (> 1 year)

TUTORIALS LOGIN FOR SUCCESS

• Slightly opaque with dark Blood vessels  Fig: 7	• Proliferative Vitreoretinopathy changes present  Fig: 8

Difference Between types of RD

	RRD	TRD	EX RD
Pathogenesis	Dynamic VR traction Acute PVD	Static VR traction, retina pulled off	Pump failure
Etiology			
Appearance	Bullae/fold / undulating/ till ora/convex	Concave	May be central /peripheral gravity dependent

Tear	Break present	Secondary break can occur	–
Subretinal fluid	Clear	Clear	Turbid
Symptoms			

Management of RD 00:32:57

- For Exudative RD – Treat the cause
- Simple RRD: – Visible tear
  - External surgery
    - 1) PneumoRetinopexy
      - i. OPD procedureretina can be sealed using laser photocoagulation
      - ii. Only in superior Retinal tears
      - iii. SF6/C3F8 used
    - 2) Scleral Bucking Surgery

DC TUTORIALS
LOGIN FOR SUCCESS

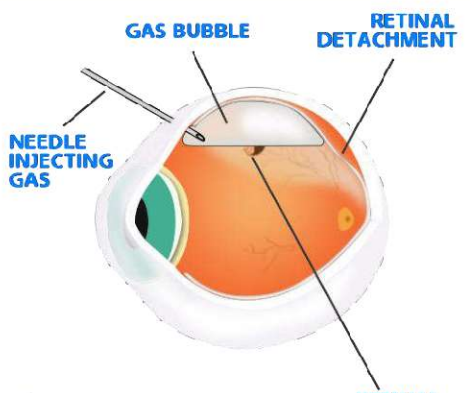

GAS BUBBLE
RETINAL DETACHMENT
NEEDLE INJECTING GAS
RETINAL TEAR

Fig: 9

Note → Lincoff's Rule

- Gives information about location of tear based on configuration of RD

Sclear Buckle

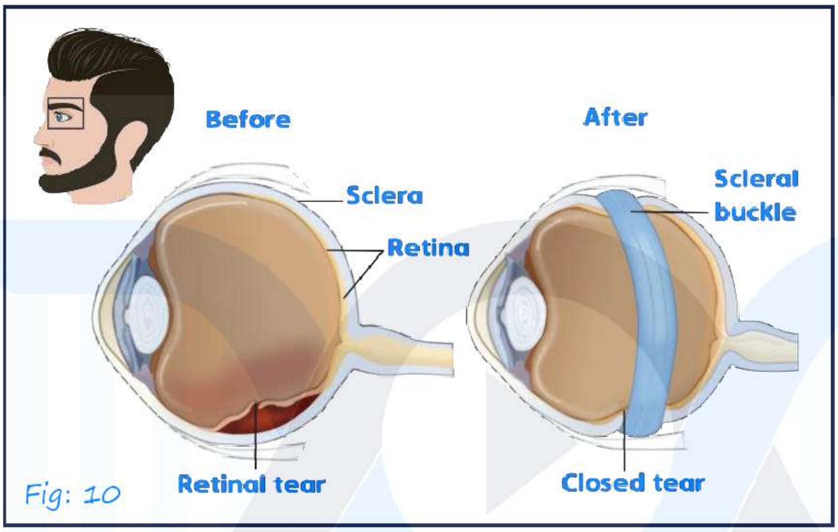

Before
After
Sclera
Retina
Scleral buckle
Retinal tear
Closed tear

Fig: 10

- Placement of silicon scleral Buckle externally over retina – where tear is present

+

360degrees encirclage

+

SRF Drainage

+

Laser photocoagulation

In RRD where tear is not located /TRD

- 3 port pars plana vitrectomy

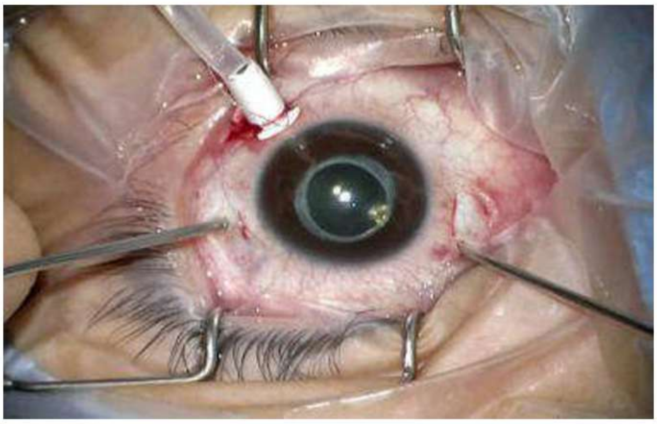


Fig: 11

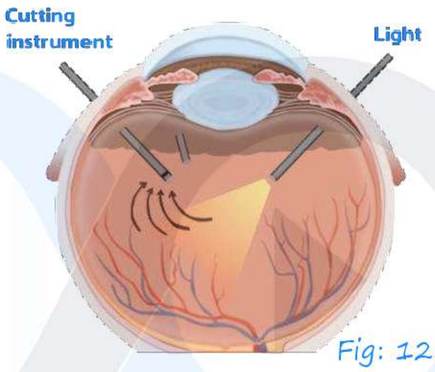


Fig: 12

↓

i. Endo illuminator

ii. Cutting instrument/vitrectomy cutter

iii. Infuser -for fluid infusion

- After vitrectomy, endo photocoagulation (laser Barrage) to seal the tear is done +

Sub Retinal Fluid Drainage

+

Tamponading agent is inserted

Tamponading agent

Gas	Silicone oil
SF6 C3 F8 • Shorter acting Longer acting • Absorbed after Absorbed after • 4-6 weeks 6-8 weeks Advantage: • Self-absorbing Disadvantage: • Air travel contraindicated since decrease pressure causes Expansion of gas & It can lead to intractable glaucoma • Positioning of pt. for proper tamponade effect is required • Visual recovery is slow (Due to interference of gas in visual axis)	Advantage: • Early visual recovery • Air travel permitted Disadvantage: • Removal of oil is needed with another surgical procedure after 3 months • Risk of tear opening • Emulsification of silicon oil can occur ↓ Its entry to AC via zonular Defect gives appearance of inverse Hypopyon (since it is lighter than aqueous ↓ It can block trabecular mesh Work ↓ IOP increases (glaucoma) ∴ it is Drained A/K/A: Hyperoleon

DOC TUTORIALS LOGIN FOR SUCCESS

Fig: 14

Typical lattice Degeneration 00:47:24

- Spindle-shaped Island of Retinal Thinning in Mid periphery of Retina
- Network of crisscross /Arborizing white lines within islands is seen
- Overlying vitreous liquefiaction seen

  ∴ increase Risk of Retinal Tear
- Can be seen in 8-10% of normal people
- More common in pathological Myopia
- 40% of pt. with RRD lattice Degeneration

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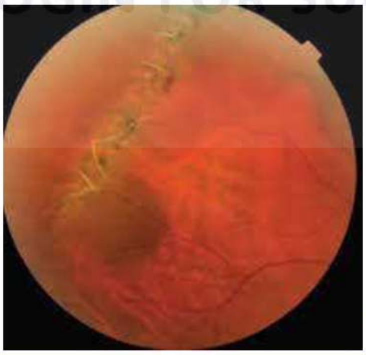


Fig: 15

Treatment

- Indication: lattice Degeneration+ Tear /sudden onset of floaters & flashes

↓

Laser Barrage (double frequency ND yag /Argon laser)

"It is to prevent Tears formation and RRD"

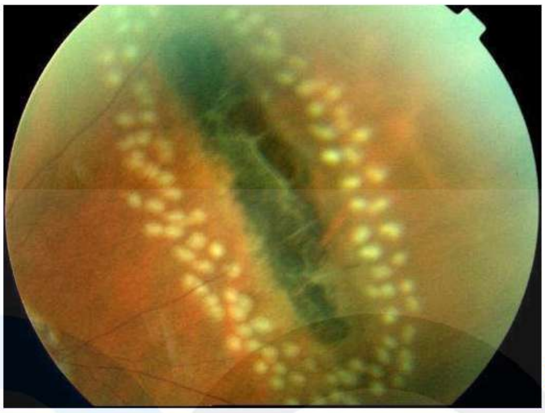


Fig: 16

Snail Track Degeneration

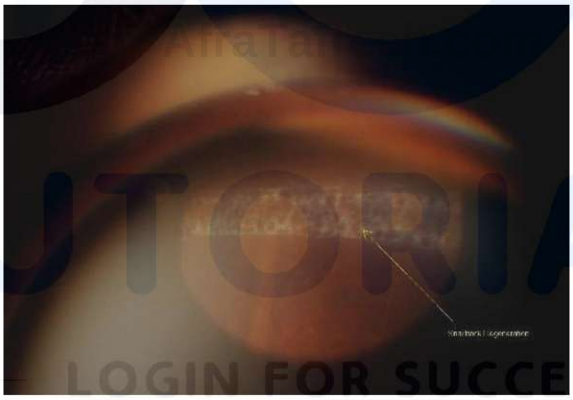

Snail track Degeneration

Fig: 17

- Sharply demarcated, frost-like bands which are longer than lattice

White without Pressure

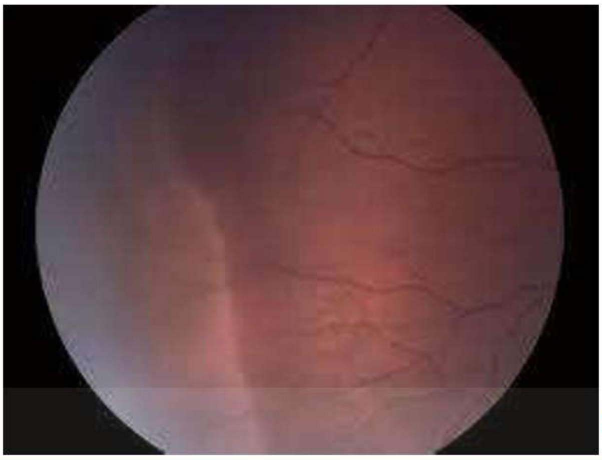


Fig: 18

- Translucent grey appearance of retina

DIABETIC RETINOPATHY

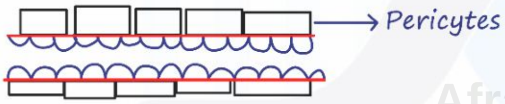
Risk factor

- Duration of diabetes (most imp)
- Uncontrolled DM
- Presence of hypertension & Nephropathy
- Pregnancy

Mechanism 00:02:42

Microangiopathy

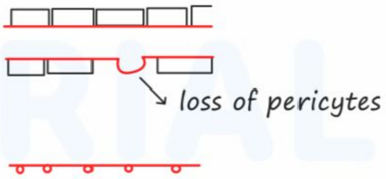
- Alter capillaries



Normal structure

In diabetes

Focal loss of pericytes occur
(1st clinically significant sign)
↓
Focal outpouching from
capillary wall (Microaneurysm)



- Microaneurysms present in inner nuclear layer
- <63μm in size

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Hemorrhages

- Flame shaped hemorrhages or superficial hemorrhages in nerve fibre layer
- Seen at periarteriolar capillary level
- Most common in **hypertensive retinopathy**

- Dot & Blot hemorrhages
- Seen in outer plexiform & inner nuclear layer
- Deeper hemorrhage
- Most common in **diabetic retinopathy**

Exudates

- Due to ischemia associated leakage
- Lipid exudation from capillary walls seen (Hard exudates) -Yellowish, small well-defined structures
- FFA – Hypo Fluorescent area near exudates
- These are seen in outer plexiform layer (it is water shed area high risk of fluid accumulation) → Also known as Henle's layer at macula

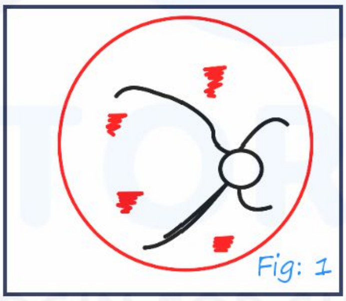

Fig: 1

TUTORIALS LOGIN FOR SUCCESS

Soft exudate

- Cotton wool spot – Pale large fluffy ill- defined areas
- Histologically called cytoid bodies
- Ischemia of NFL → Causes stasis in axons → Appear as cotton wool spots → Hyperfluorescent on FFA

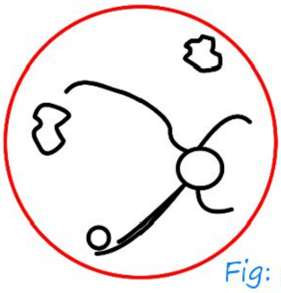

Fig: 2

Venous changes

- Venous beading
- Venous looping

Intra retinal microvascular abnormalities (IRMA'S)

- There are anterior – venous shunts – Abnormal vessels

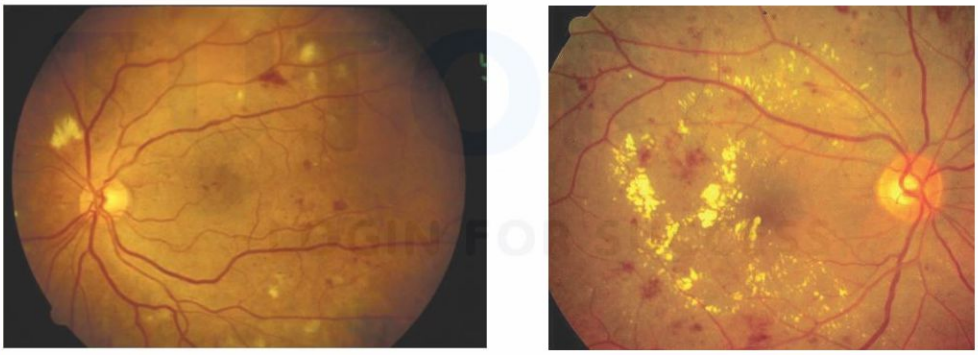

Fig: 3

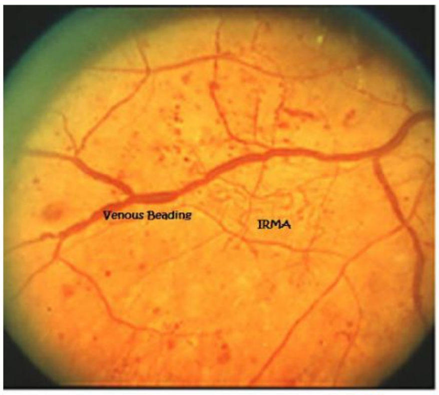

Fig: 4

ETDRS-Early treatment diabetic retinopathy study 00:16:16

Form/Category	Characteristic
Non proliferative diabetic retinopathy (NPDR)	
Mild NPDR	Microaneurysms only
Moderate NPDR	More than just microaneurysms but less than severe NPDR
Severe NPDR 4-2-1 rule	Any of the following and no signs of proliferative retinopathy. Severe intraretinal hemorrhages and microaneurysms in each of 4 quadrants or Definite venous beading in 2 or more quadrants or Moderate IRMA in 1 or more quadrant
Very severe NPDR	≥2 criteria for severe NPDR in the absence of frank neovascularization
Proliferative diabetic retinopathy	
PDR	One or both of the following • Neovascularization • Vitreous preretinal hemorrhage

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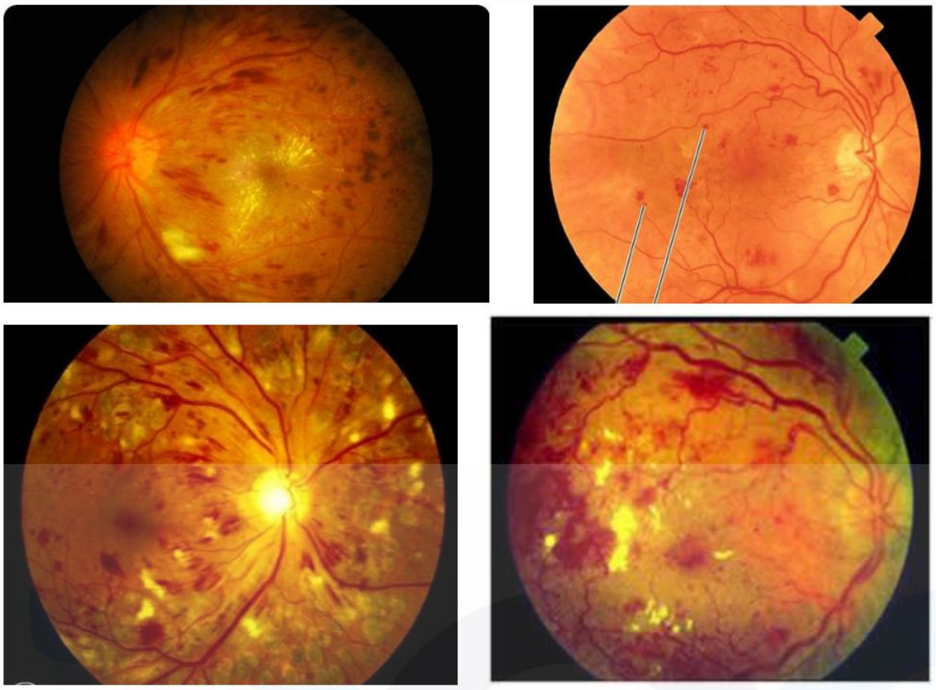


Fig: 5

Proliferative Diabetic Retinopathy

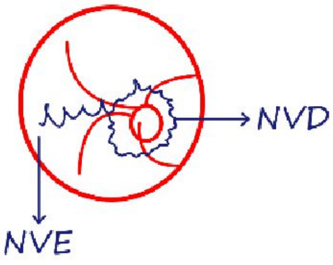
PDR

NVD

- Neovascularization at disc and also within 1DD area around disc

NVE

- Neovascularization elsewhere



High risk PDR

- Definition:new vessels on disk (NVD) >1/3 ODD with/Without hemorrhage (preretinal & vitreous)
- More dangerous
- NVE
- >1/2 ODD with/Without hemorrhage
- <1/2 ODD with hemorrhage (Preretinal & vitreous hemorrhage)
- Immediate photocoagulation

Macular edema 00:25:40

- Collection of hard exudates with retinal thickening
- Causes:
  - i. Leakage from a microaneurysm (Focal ME)

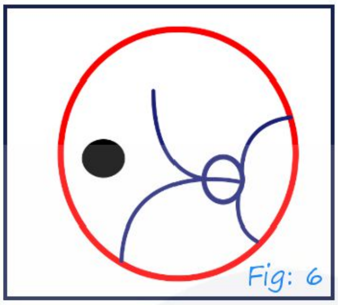


Fig: 6

  - ii. Diffuse leakage due to breakdown of inner blood retinal barrier called as diffuse ME

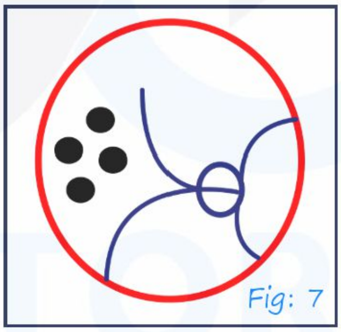


Fig: 7

Clinically significant ME

- 3 criteria
  - Any retinal edema within 500µm of the center fovea
  - Hard exudates within in 500m of the center of the fovea if associated with adjacent areas of retinal thickening
  - Retinal edema >1 disc area in size & within 1disc diameter of center of fovea

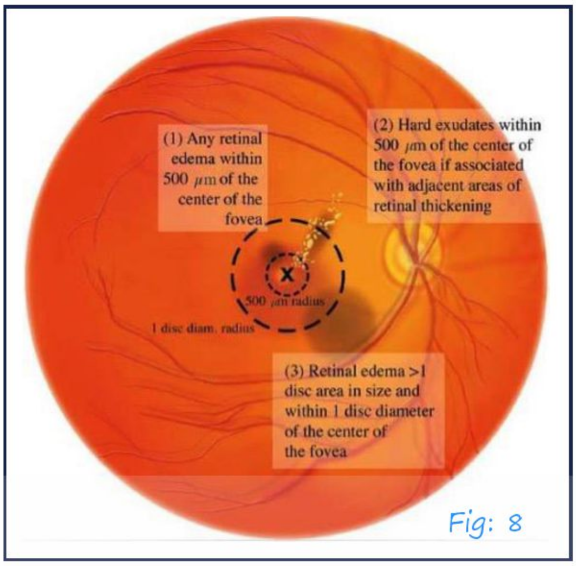

(1) Any retinal edema within 500 μm of the center of the fovea
(2) Hard exudates within 500 μm of the center of the fovea if associated with adjacent areas of retinal thickening
X
500 μm radius
1 disc diam. radius
(3) Retinal edema >1 disc area in size and within 1 disc diameter of the center of the fovea

Fig: 8

Diagnosis

- Ophthalmoscopy
- Best method: Slit lamp bimicroscopy (Examination on slit lamp with in 90D lens)
- OCT (Optical coherence tomography)

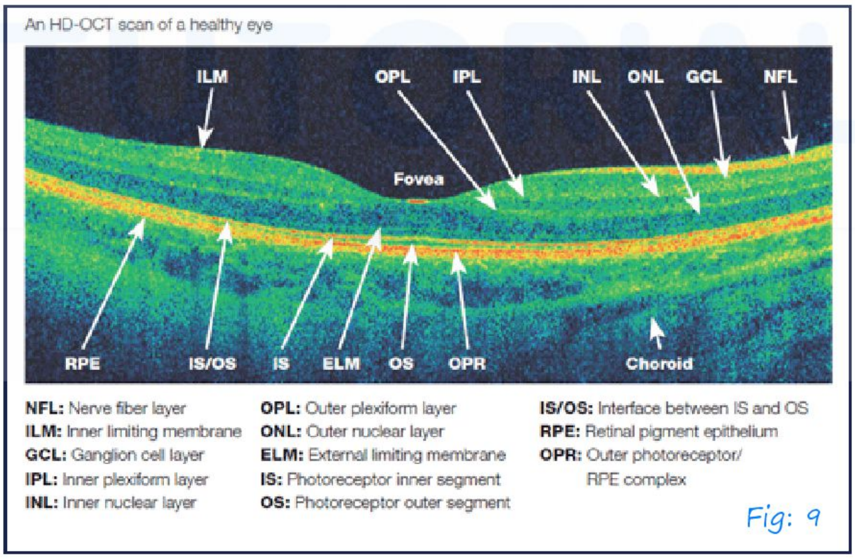

An HD-OCT scan of a healthy eye
ILM
OPL
IPL
INL
ONL
GCL
NFL
Fovea
RPE
IS/OS
IS
ELM
OS
OPR
Choroid
NFL: Nerve fiber layer
ILM: Inner limiting membrane
GCL: Ganglion cell layer
IPL: Inner plexiform layer
INL: Inner nuclear layer
OPL: Outer plexiform layer
ONL: Outer nuclear layer
ELM: External limiting membrane
IS: Photoreceptor inner segment
OS: Photoreceptor outer segment
IS/OS: Interface between IS and OS
RPE: Retinal pigment epithelium
OPR: Outer photoreceptor/ RPE complex

Fig: 9

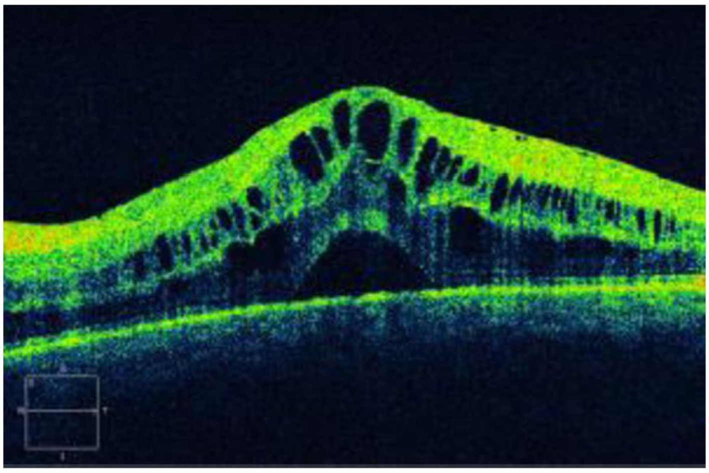


Fig: 10

Treatment

- For focal ME: Focal laser
- For diffuse ME: Anti VEGF Injection & Grid laser
- "Spot size is less & Does not cause visual field defects"

Causes of decrease in vision in DR 00:33:00

- Most common cause is macular edema
- Most common cause of sudden decrease in vision
  - Vitreous hemorrhage (Most common)
  - Tractional retinal detachment → It can cause slow decrease in vision also
- Most common cause decrease in vision in DM – Cataract

Treatment of DR 00:35:20

When to treat DR?

- If NPDR + CME

- If PSR – Especially HR-PDR – Urgent treatment

Note

- In very severe NPDR, PDR if Patient has high risk factors & Difficulty for follow up, he can be treated "In clinical setting)

For NPDR follow up is needed

- Mild – Every 9months
- Moderate – Every 6 months
- Severe – Every 3 months
- Very severe – Every 2 months

Educate: Strict DM control

Screening

- For type 1: After 5yrs of diagnosis
- For type 2: As soon as possible

Treatment

- Anti VEGF's

  a. Bevacizumab (Avartin)

    - Full sized recombined humanized monoclonal antibody
    - It blocks VEGF-A
    - Dose: 1.25mg /0.05ml – Intravitreal injection

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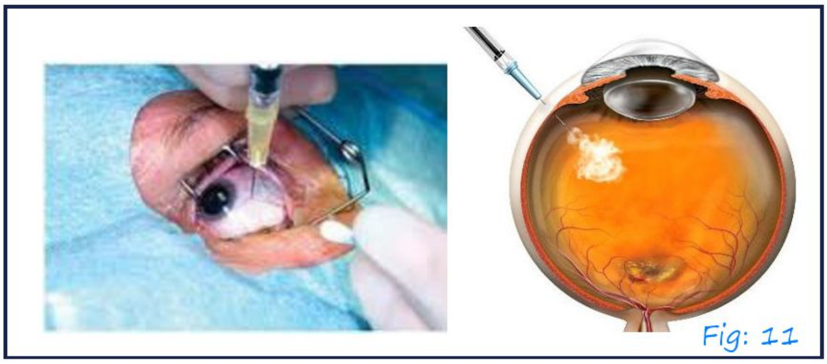


Fig: 11

b. Ranibizumab
  - FDA approved
  - Monoclonal AB fragment
  - It has better binding capacity
  - Inhibit all isoforms of VEGFA
  - 0.5mg/0.05ml

c. Aflibercept – Also known as – Eye lea/ VEGF trap / Decoy receptor
  - Recombinant fusion protein – VEGF binds it instead of its receptors

d. Pegaptanib – Macugen
  - It is aptamer for VEGFA 165
  - Selective action
  - Mainly used in wet age-related macular degeneration

- Intravitreal steroids
  - Triamcinolone – Long-acting steroid
  - Dexamethasone implant (OZURDEX) → Sustained

- Laser photocoagulation
  - Pan retinal photocoagulation: Laser throughout retina except macula & optic disc

↓ It has papulo macular bundle

↓ Ganglion cells

Central scotoma

- Laser photocoagulation converts hypoxia → Anoxia (Dead tissue)

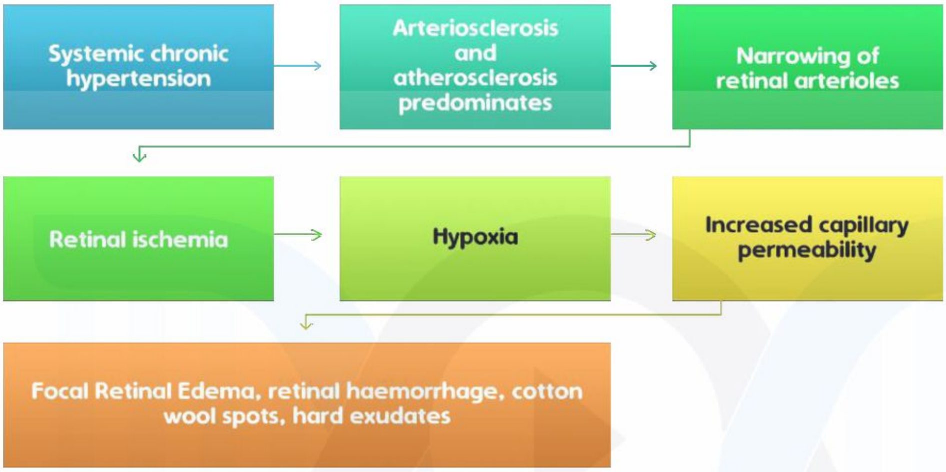
Anoxia ↓ Peripheral retina → Oxygen given to imp central retinal thus hypoxia is corrected & VEGF not released

- Laser: Argon green laser/Double frequency Nd YAG laser (532mm) (510nm)
- Side effects: visual field defect & Gradual decrease of visual field occurs
- 1500-1800 yellow spots given
- Done in 3-4 sittings
- To check for visual field defects
- To prevent severe inflammation & Glaucoma
- First inferior retina lasered
- Laser power: 300-500
- Duration of laser: 0.1sec
- PRP is treatment of choice for PDR & HRPDR
- If tractional & Element is present – Laser is contraindicated

HYPERTENSIVE RETINOPATHY

Pathophysiology 00:00:39

- Here the problem is related to vasospasm


Systemic chronic hypertension
Arteriosclerosis and atherosclerosis predominates
Narrowing of retinal arterioles
Retinal ischemia
Hypoxia
Increased capillary permeability
Focal Retinal Edema, retinal haemorrhage, cotton wool spots, hard exudates

- Clinical presentation includes
  - Retinopathy → in chronic hypertensives
  - choroidopathy → Acute accelerated HTN, mainly in young patient
  - Optic Neuropathy

Clinical features 00:02:40

Keith-Wagner- Barker classification

- Group 1: Slight constriction of retinal arterioles
- Group 2: Group 1 + focal narrowing of retinal arterioles + AV nicking- Salu`s sign

- Group 3: Group 2 + flame-shaped hemorrhages + cotton-wool spots + hard exudates- BONNET SIGN AND GUNN SIGN: Copper wiring
- Group 4: Group 3 + optic disc swelling- Silver wiring +macular star formation

For Hypertensive Retinopathy

- Stage 1: Diffuse arteriolar narrowing
- Stage 2: Stage 1 + focal arteriolar constriction
- Stage 3: Stage 2 + retinal hemorrhage
- Stage 4: Stage 3 + hard exudates + retinal edema+ optic disc swelling

For Arteriosclerosis

- Stage 1: Broadening of arteriolar light reflex
- Stage 2: Stage 1 + AV crossing changes
- Stage 3: Copper wiring of arterioles
- Stage 4: Silver wiring of arterioles

TUTORIALS LOGIN FOR SUCCESS

Keith Wagner Barker

- Anterior narrowing /spasm

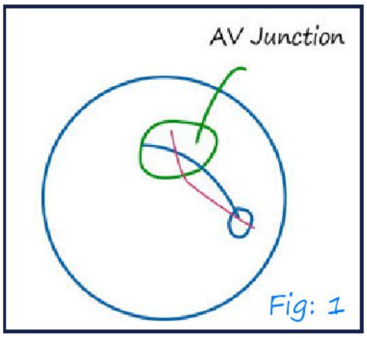


Fig: 1

- A:V ratio → 2:3

  but in arteriolar spasm → 1:3

At AV junction: - Salu's sign

- 'S' shaped deflection of vein

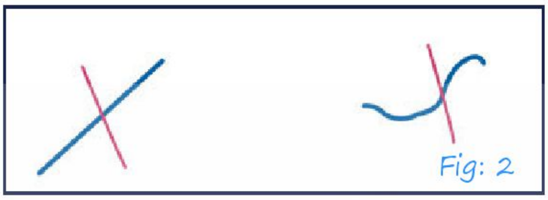


Fig: 2

- we can also see Hemorrhage, exudates at this stage

Bonnet sing – banking – dilation

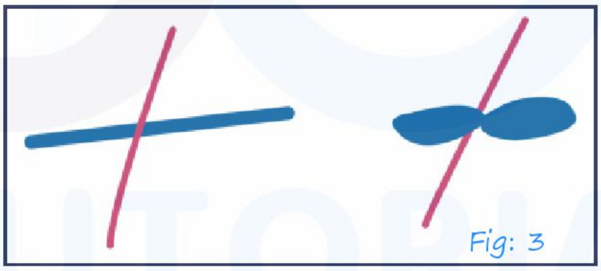


Fig: 3

Gunn sign

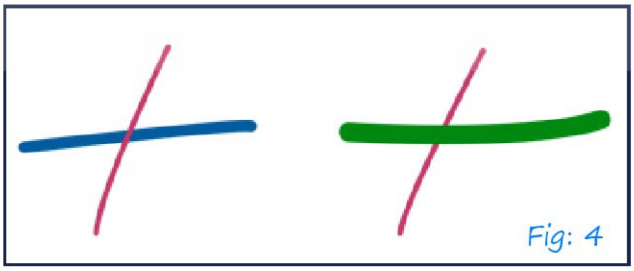


Fig: 4

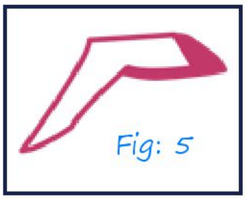


Fig: 5

- Copper wiring appearance
- Copper wire arterioles where the central light reflex occupies most of the width of the arteriole

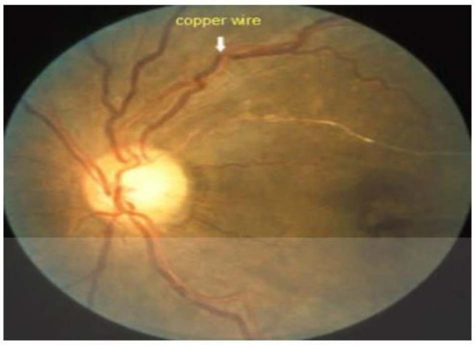


Fig: 6

Silver wiring

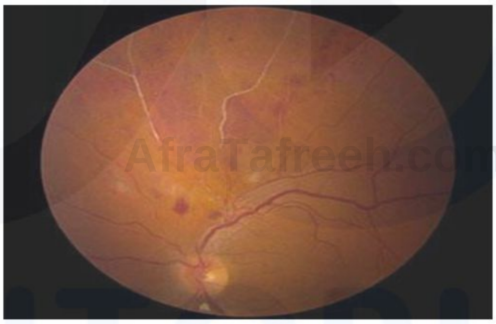


Fig: 7

B/L Papilledema + macular star + silver wiring

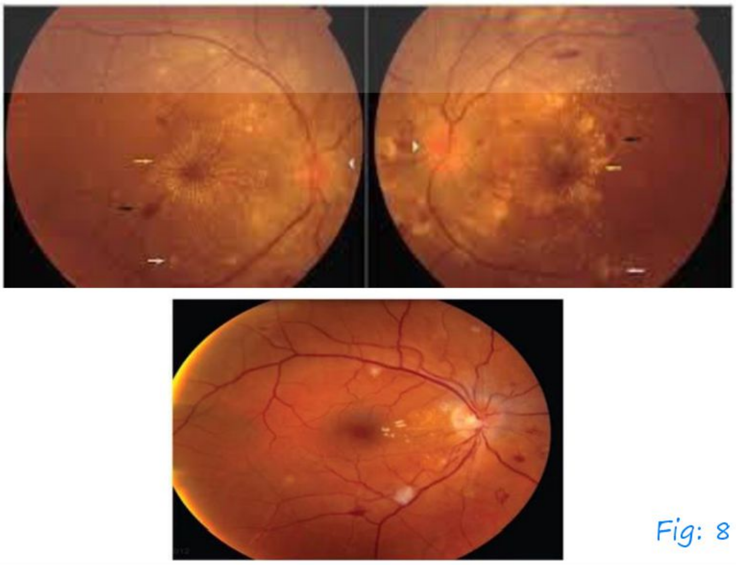


Fig: 8

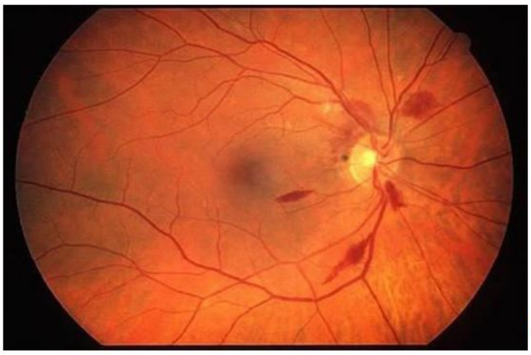


Fig: 9

Choroidopathy 00:11:20

Hypertensive Choroidopathy

- Typically occurs in young patients
- Episode of acute hypertension associated with preeclampsia, eclampsia, pheochromocytoma, or renal hypertension
  - Elschnig spots – non perfused choriocapillaris
  - Siegrist streaks – hyperpigmented streaks

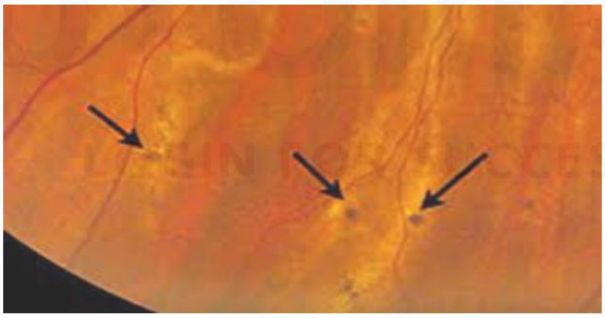


Fig: 10

- Siegrist streaks are described as hyper-pigmented flecks that are arranged in a linear fashion along the choroidal vessels of the eye

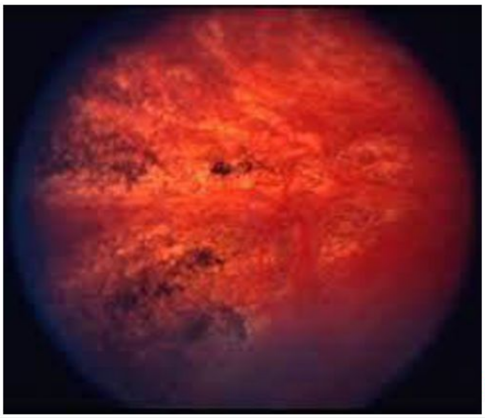


Fig: 11

- Fibroid necrosis of choroidal vessels

Hypertensive optic Neuropathy 00:13:10

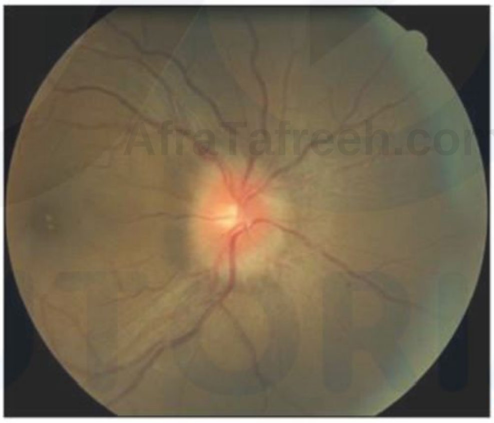


Fig: 12

- Vasculitis

RETINAL VASCULAR OCCLUSIONS

Central Retinal Venous Occlusion [CRVO]

00:00:10

1.

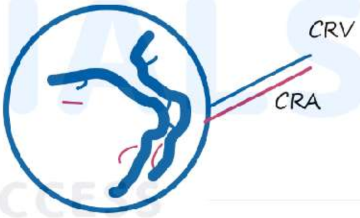
- Compression of central retinal vein by central retinal artery (most common)
- It is seen in conditions like atherosclerosis, HTN, Hyperlipedemia, open angle glaucoma, DM & In elderly

2.Thrombotic CRV lumen

3.Inflammation of CRV

Types based on presentation

1.Non ischemic = venous stasis CRVO

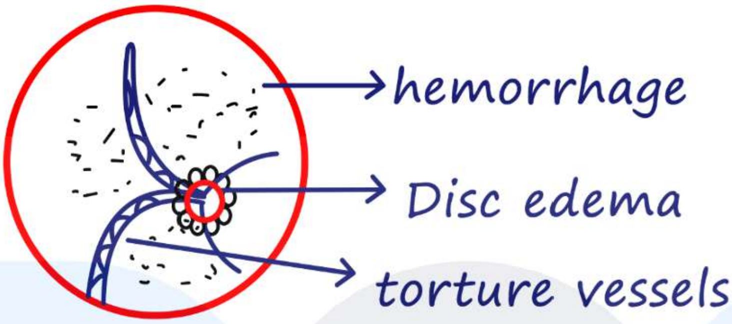


- Decreases vision > 6/60
- Pupils normal
- Veins tortuous, dilated
- Macular Edema (Due to hypoxia induced leakage) change
- Few flame shaped hemorrhage

2.Ischemic CRVO = hemorrhagic/fast progressive CRVO

- Incidence 20-30%
- Hypoxia & Ischemia → New vessels → Bleed

- RAPD due to disc edema
- Sudden gross decrease in vision < 6/60
- Tomato splash appearance/ketchup appearance (due to hemorrhage)



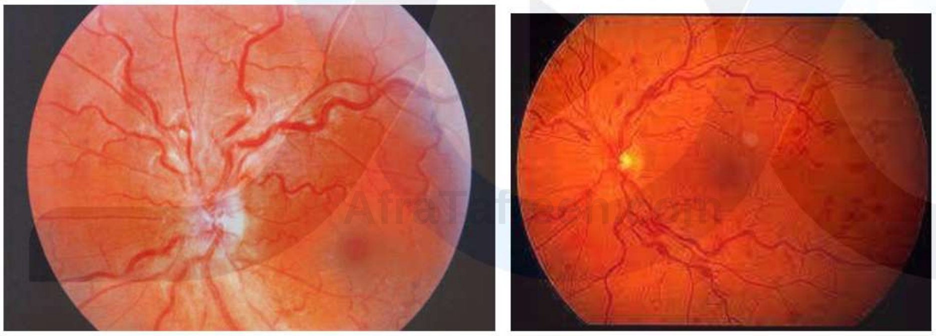

CRVO – Non Ischemic

Fig: 1

Ischemic CRVO/Splashed Tomato/Thunderstorm

- Retina pooled with hemorrhages.
- High risk of neovascularization due to severe ischemia

↓

It can further lead to neovascularization of anterior chamber

↓

Patient may develop 2° neovascular glaucoma

A/K/A – 100-day glaucoma

- Note – Most common cause of neovascular glaucoma is DM

	Non Ischemic CRVO	Ischemic CRVO
	Venous Stasis	Hemorrhagic
	Slow process	Fast process
Percentage	75%	25% (non ischemic can get converted into ischemic)
Retina appearance & presentation	Venous tortuosity, hemorrhage, exudates	Splash tomato appearance
Macula	Macular edema ⊕	Severe macular edema & macular ischemia
RAPD	Absent	Present
FFA	<5DD area of non-perfusion	> 10DD area of non- perfusion
Prognosis	Good	Poor (Due to macular ischemia)

Treatment

- Hemorrhages resolves on its own
- Macular edema + non ischemic CRVO → Anti VEGF is drug of choice

  Laser treatment is not approved

  Macular ischemia + ischemic CRVO → Intravitreous steroids.

Branch retinal venous occlusions

- Localize hemorrhages
- Most common location – supero temporal

  ↓

  Most common BRVO to be diagnosed

  ↓

  (Decreases vision) – patient presents to the hospital with this complaint
- It is more common than CRVO

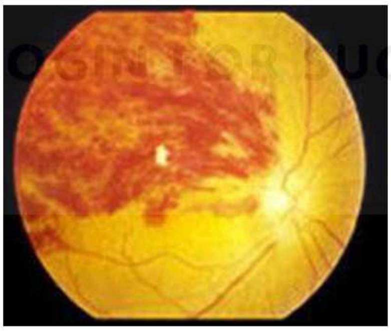


Fig: 2

Ischemia

↓

New vessels

↓

In BRVO more NVE is seen than NVI

Treatment

- Laser → Better
- Anti VEGF

Hemi retinal venous occlusion

- Problem at level of retinal trunk

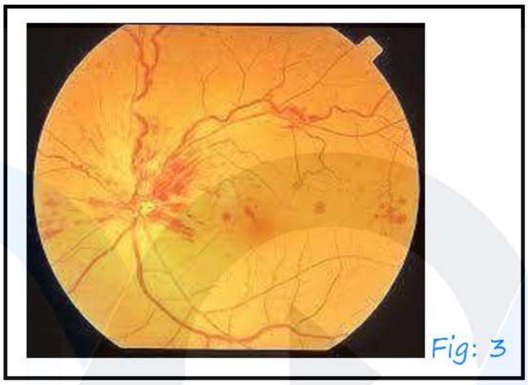


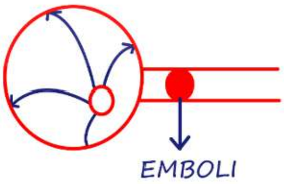
Fig: 3

Papillophlebitis 00:16:50

- CRVO of young's
- Vasculitis caused by inflammatory pathologies

Central Retinal Artery Occlusion 00:15:40

TUTORIALS LOGIN FOR SUCCESS



- Most commonly caused by embolism

↓

It may originate from common carotid → internal carotid

↓

Opthalmic artery

- Hollen horst plaque → cholesterol filled embolus

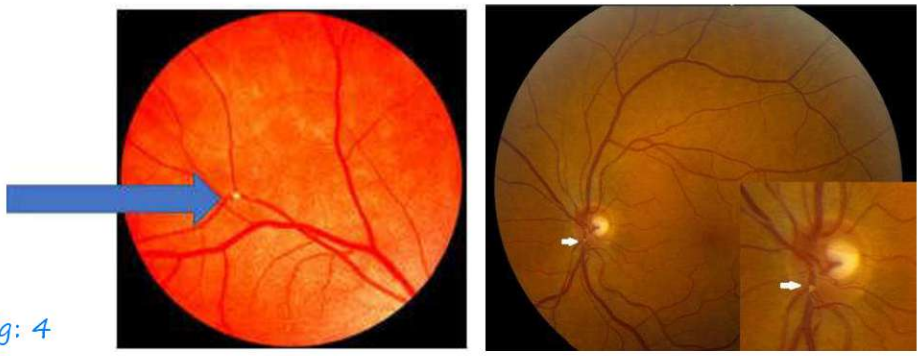


Fig: 4

- Calcific plaque → most dangerous
- Platelet fibrin

Clinical feature

- Sudden painless loss of vision

  In giant cell arteritis – pain may be there.

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Sign

1. Whitening of Retina

   CRAO → Ischemia at level of ganglion cell layer → edema

↓

Retina loose it's transparency & it becomes white.

↓

Resolve After few months – normal retina seen.

CRAO with Cilio retinal Artery 00:29:08

Here only macular area is spared due to cilioretinal artery

↓

It is present in 20%-30% of normal individual

It is branch from posterior ciliary artery

- Because of this sparing vision is spared.

- Cilioretinal artery occlusion: central area becomes pale, rest of retinal is normal in appearance.

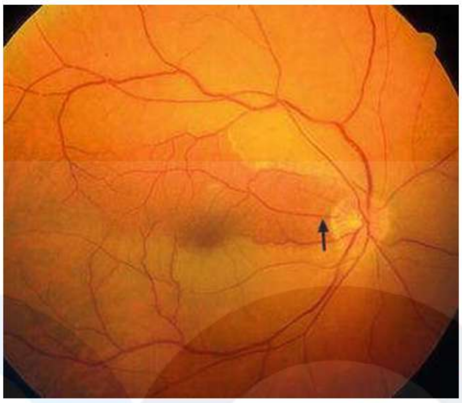


Fig: 5

- It can present with CRVO/CRAO

Differential Diagnosis of Cherry Red Spot

00:31:42

- Mnemonic: cherry tree never grow tall in mud & sand
  - C – CRAO, CO-poisoning
  - T – Tay sach's disease (GM_2 gangliosidosis seen in 95% patient with tay sach's)
  - N – Neimann pick
  - G – Generalized Gangliosidosis (GM1 Type1)
  - T – Trauma Blunt/ Berlin edema
  - M – Metachromatic leukodystrophy, Methanol poisoning
  - S – Sandoff's disease, sialodosis
- Type 2 gauchers only shows cherry red spot

TUTORIALS LOGIN FOR SUCCESS

2.Cherry red spot: Bright red spot at fovea.

∵ Fovea do not have ganglion cells – edema is absent & thus it remains normal.

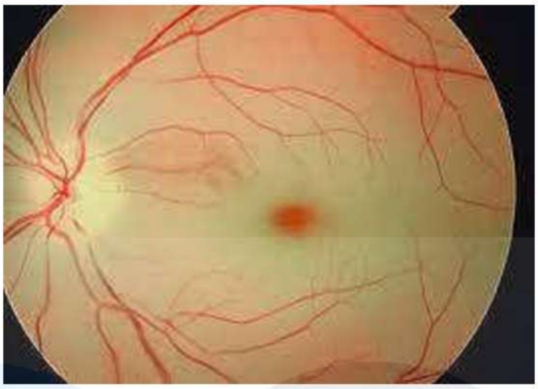


Fig: 6

3.Cattle trucking sign/Box carrying sign:

↓

It is due to segmental blood flow caused by emboli

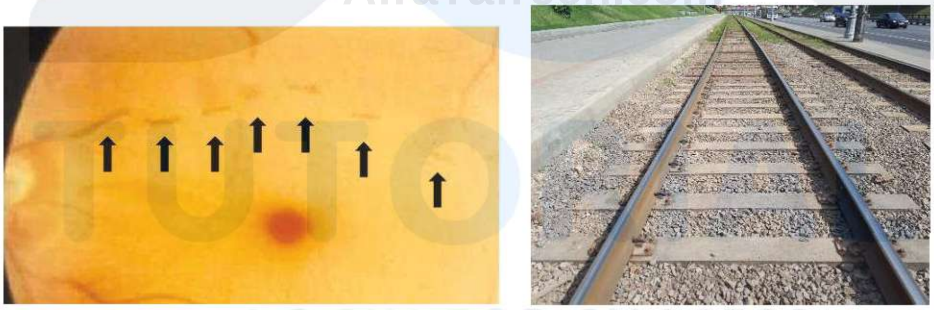


Fig: 7

4.RAPD ⊕

Management

1.Vigorous ocular massage: embolus may dislodge

2.Vasodilation

- Sublingual isosorbide
- Calcium channel blockers

- Carbogen (95% O_2 + 5% CO_2)

3.Decrease IOP:

- Mannitol
- Acetazolamide
- Paracentesis

4.Intra arterial streptokinase

Note

- Poor prognosis: salvageable window time is 100 min ∵ beyond 100 min ganglion cell layer gets damaged.
- ∵ painless patient present late to doctor.

EALES DISEASE

- Idiopathic peri-phlebitis

  ↓

  Inflammation of vessels- veins > arteries
- Association of tuberculin Hypersensitivity
- Mostly seen in young males- 2nd/3rd decade

Stages 00:01:42

Fig: 1

- Inflammation → Floaters

  → Re- steroids
- State of occlusion (or) Ischemia → Mx-FFA

  → Sectoral laser

  → Anti VEGF
- Complication/Neovascularization

  after Neovascularization there can be belling leading to vitreous

  hemorrhage

  ↓

  sudden painless decease in vision

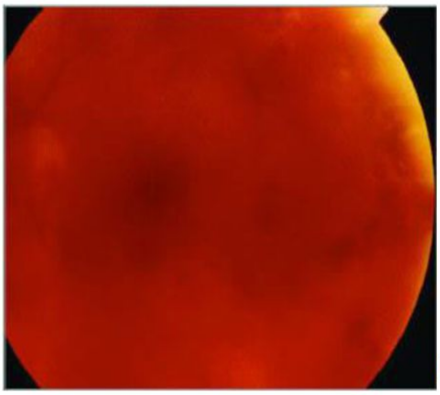


Fig: 2

- Boat shaped/sub hyaloid hemorrhage

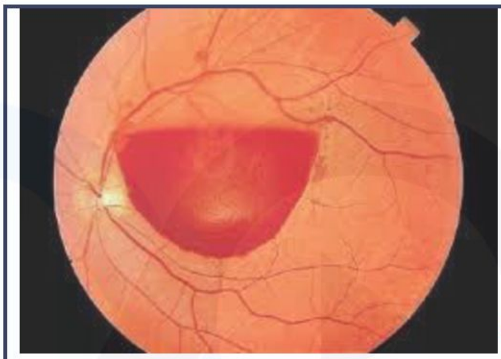


Fig: 3

- In case of vitreous hemorrhage

  Mx- wait and watch

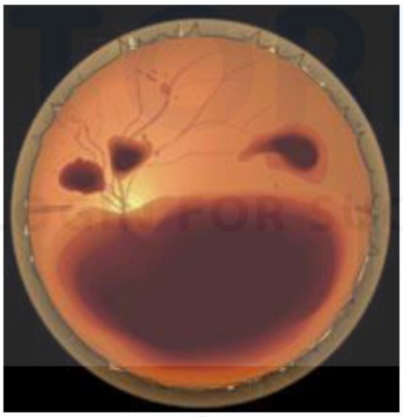


Fig: 4

↓

Hemorrhage clears

↓

FFA- to find the leak /areas of Ischemia

↓

PRP

- If patient has tuberculin hypersensitivity/TB as underlying cause then start ATT

MACULAR DISORDER

Cystoid Macular Edema 00:00:24

cystoid macular edema

- Retinal thickening + Hard exudates in retina

↓

Occur at level of outer plexiform layer

- It occurs due to breakdown of inner blood retinal barrier

Causes

- Drip or nose – mnemonic
- D → Diabetic retinopathy
- R → Retinitis pigmentosa
- I → Irvine gas syndrome
- P → Prostaglandin analogue
- Ur → Uveitis
- N → Niacin
- O → Occlusion CRVO, BRVO
- S → Surgery
- E → Epinephrine – Cause CME in aphakic pt

Clinical features

- Decreased in central vision

- Micropia (Smaller imager of object) → Due to fluid collection photoreceptors get separated leading to formation of smaller objects
- Metamorphopsia → distorted imager
- On slit lamp biomicroscopy with 90° D lens honey comb pattern of macula is seen

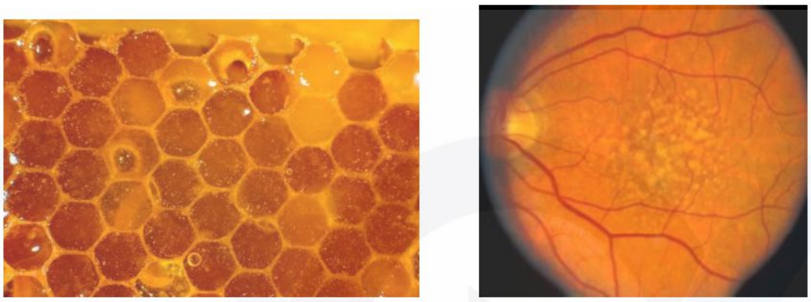


Fig: 1

- Petaloid appearance is seen in FFA

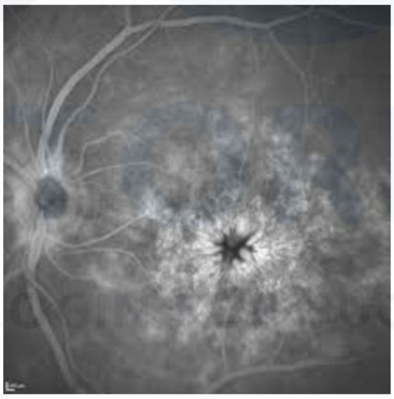


Fig: 2

- It is due to pooling of dye
- It typically occurs dur to Henle's layer (Outer plexiform layer at macula)
- OCT for quantification type of edema
- Cystoid pattern edema is seen
- It is due to necrosis of muller cells

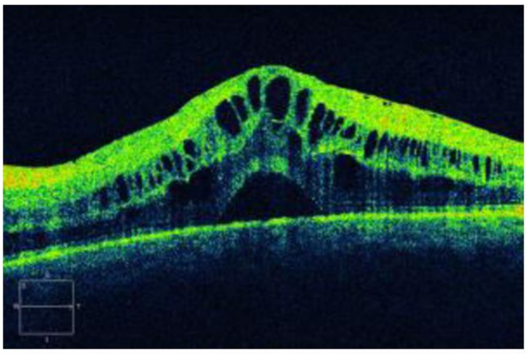


Fig: 2

Treatment

- Anti VEGF

Central serous retinopathy 00:06:30

Central serous retinopathy

- Central → Macular
- Serous → Fluid
- It is a type of focal exudative macular detachment
- It occurs due to hyperpermeability of choroid/RPE → Collection of fluid in subretinal space

Etiology

- Commonly seen in young male, Type A personality, aggressive, ambitious, stress, smoker
- H. pylori infestation
- Steroid intake

Symptoms

- Decreased in central vision (It is due to induced hypermetropia)

- Due to collection of fluid in retina it moves forward & thus light rays fall behind retina
- Central scotoma is seen – In CSR positive scotoma is seen
- Note: In glaucoma: Negative scotoma is seen

Signs

- Halo reflex is seen
- FFA shows in enlarging dot sign
- It is most common finding

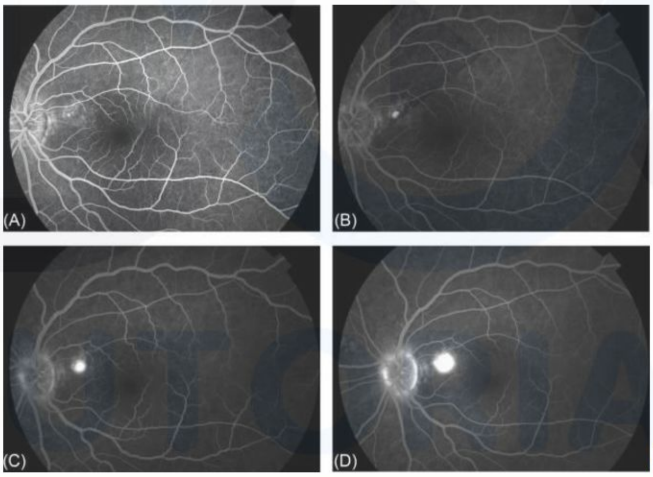


Fig: 3

- Mushroom pattern/Umbrella pattern/smoke stack pattern

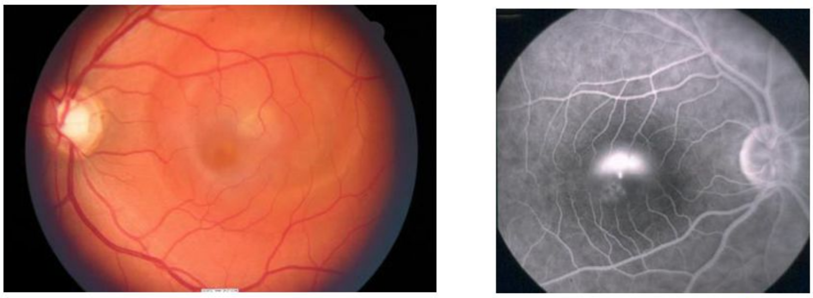


Fig: 4

OCT

- Fluid collection in subretinal space – Neurosensory detachment

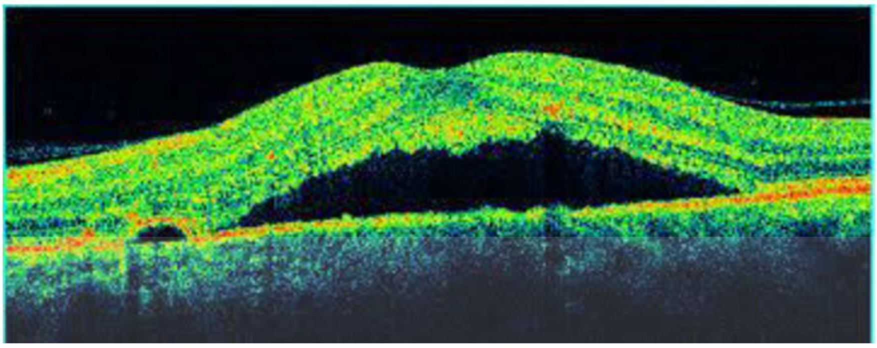


Fig: 6

Note

- In pigment epithelium detachment – Fluid is collected in sub RPE space

Treatment

- It is self-limiting → Resolve in 2-3 months minimize risk factors life style changes
- Anti VEGF injection
- PDT – Photodynamic therapy

In chronic CSR
Bilateral CSR

Macular Hole 00:19:10

Macular hole

- Full thickness defect in macula
- Most cases are idiopathic
- Mostly seen in female & Elderly
- H/O exposure to UV light present
- After lasers

- Cystoid macular edema
- Clinical features -central vision is decreased

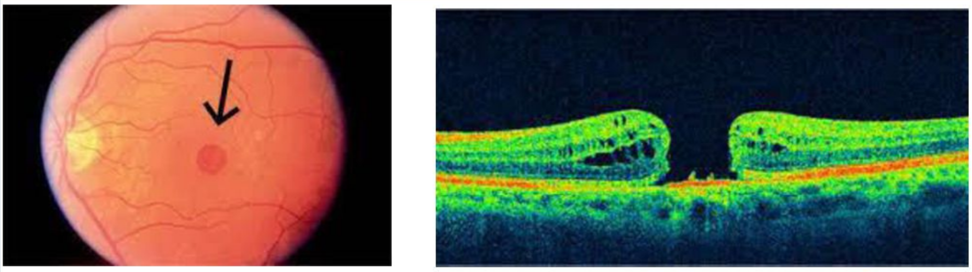


Fig: 7

Note

- In lamellar hole → photoreceptors are intact

Treatment

- If large full thickness hole is present than
- Pare plana vitrectomy + ILM peeling + Gas in vitreous cavity

GASS staging

- Four stages are present
- Full thickness defect starts from stage 2

ARMD (age related macular degeneration)

00:22:15

ARMD (Age related macular degeneration)

- Occurs in people >55yrs of age
- Incidence increased in people after 70yrs of age

- It is most common cause of gradual progressive painless decrease in vision in elderly in European countries

Risk factors

- Increased age
- Smoking (Due to cyanide – Damage RPE)
- More common in females
- ↑↑ Fat & Meat intake
- Sunlight

Clinical features

- Collection of waste material – DRUSEN in bruch's membrane
- PAS positive extraocular substance
- It is hall mark of ARMD
- Drusen can be seen in normal individuals
- But in ARMD → Large confluent drusen's are seen

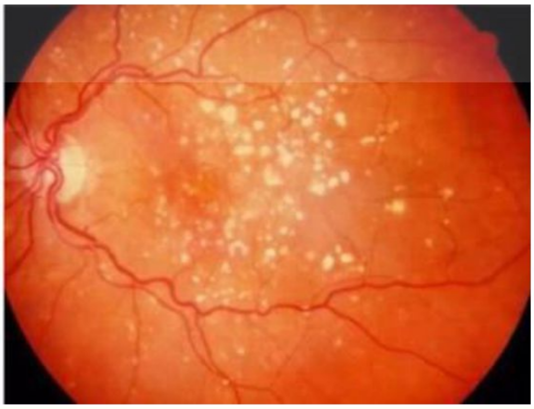


Fig: 8

Note

- Drusen: It is sub retinal therefore vessels can be seen over drusen
- Exudate: It is intra retinal therefore no vessels

Types

- Dry
- Wet

Dry

Classification	Clinical manifestation
No AMD	No drusen and no RPF abnormalities
Normal aging changes	Drusen ≤63 and no RPE abnormalities
Early EMD	Drusen >63µm and ≤125µm and/no RPE abnormalities
Late AMD	GA and/or neovascular AMD

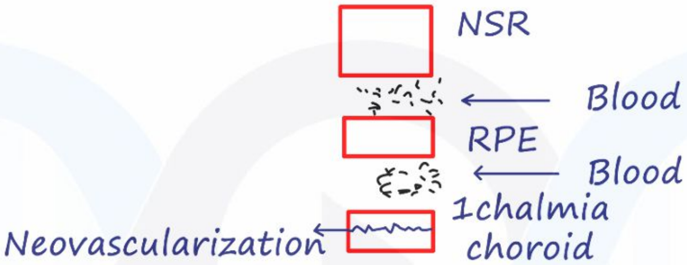
- Geographic atrophy
- Late dry AMD occurring at level of macula
- >175µm chorioretinal atrophy
- Vision lost
- Worst prognosis

Wet form

- Fluid – Due to detachment of pigment epithelium
- Blood – Coming from choroidal neovascular membrane (CNVM)

Hall mark of wet ARMD

Neovascularization at level of choroid


NSR
Blood
RPE
Blood
1chalmia choroid
Neovascularization

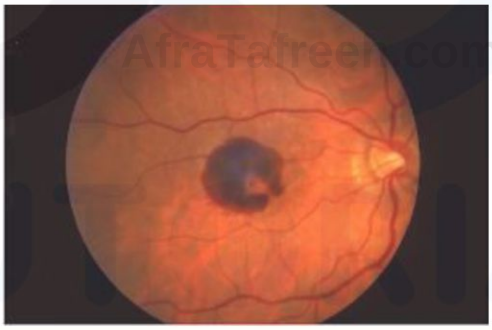
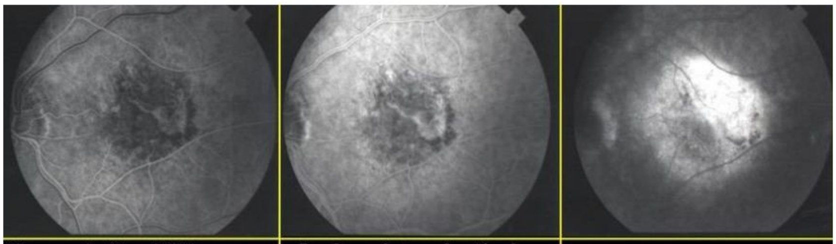


Fig: 9

- Angiography
  - Classic pattern CNVM
  - Occult pattern CNVM

Classic pattern

- Early leakage point is seen which progress to late staining



Very early 'lacy' filling pattern

Leakage into subretinal space and around CNV

Late staining

Fig: 10

Occult form

- No lacy pattern point of leakage is not seen
- ill-defined area of irregular leakage and stippled pattern seen
- ICG (Indocyanine green angiography) done
- It is derivative of fluorescein & is more protein bound (98%)
- Mostly held in choroid → It delineate choroidal pattern more

Management

- Based on AREDS-2 (Age related eye disease study)
- Treated only at intermediate stage of AMB
- Mnemonic – C2 ELZ2
- Vit C – 500mg
- Copper – 2mg
- Vit E – 400IV
- Lutein 10mg
- Zeaxanthin 2mg
- Zinc 80mg

Prevent progression to late AMD

AMSLER GRID 00:37:06

Amsler grid

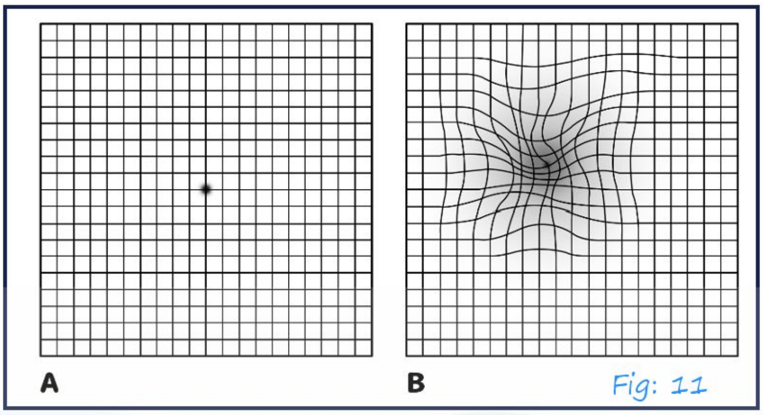


Fig: 11

- It is macular function test
- Test 20° of field from centre (Macula)
- There are 400sq of 5mm x 5m size
- Wavy pattern indicates – Disease progression at macular
- Missing field indicate – Scotoma
- It has prognosticative value

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TUTORIALS
LOGIN FOR SUCCESS

RETINAL DYSTROPHY

I. Retinitis pigmentosa 00:00:10

- Idiopathic degeneration of photoreceptors (rods > cones)
- Most of the cases are sporadic
- Most common is autosomal recessive inheritance, but AD & x linked recessive cases are also seen

AD ↓ Good prognosis

x linked recessive ↓ Worst prognosis

Clinical Features

- Nyctalopia – night blindness (∵ rods gone)
- Consecutive optic atrophy – Degeneration leads to peripheral constriction of field leading to tunnel vision.
- Gradual painless decrease in vision
- Most commonly males are affected, females are carriers
- Occurs mostly in 1st decade (or) start of second decade around 3rd decade blindness occurs.

Signs – TRIA

- B: Bony spicules (pigment left after degeneration of ROPS)
- A: Attenuated arterioles – 1st feature, most prominent feature

TUTORIALS LOGIN FOR SUCCESS

- P: pale disc
- Female shows tapetal reflex
- Pupil: shows RAPD (Relative afferent pupillary defect)

  ∵ Loss of nerve fiber layer in optic atrophy
- Complicated cataract, cystoid macular edema can be associated with this.

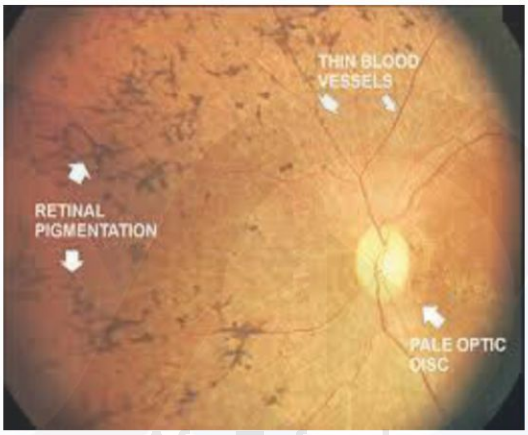


Fig: 1

- Bony spicules are maximum near mid periphery of retina∵ maximum concentration of rods is seen here.
- Examined using indirect ophthalmoscope.

Systemic association of RP (1

- (Mnemonic – BLACK URN)
- Bardet beidel syndrome
- Laurence moon beidel syndrome
- Abetalipoproteinemia (Bassen kornzweig syndrome)

  ↓

  It is associated with acanthocytes.
- Cocaine syndrome (ataxia, deafness)
- Kearn sayre syndrome

- Ushers' syndrome: sensory neural deafness present (most common association)
- Refsum (polyneuropathy)
- NARP (Narcolepsy, ataxia, retinitis pigmentosa)

Types of RP

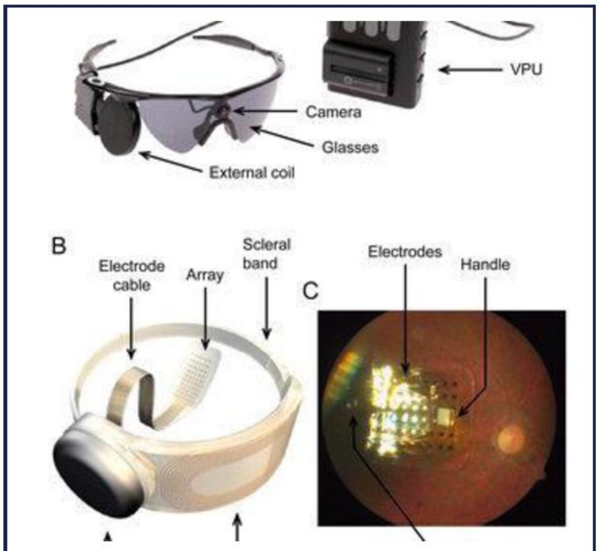
1. Inverse RP: cone rod dystrophy (cone & rods)
2. Sectoral RP: confined to single sector
3. Retinitis pigmentosa sinepigmento: Bony spicules are absent

   ↓

   - Decreases B (wave amplitude)
   - Increases implicit time
   - Finally extinguished response
4. Fundus albipunctatue – white fleck present throughout retina.

Treatment

1. Vitamin A long term prophylaxis → it will increase time of onset of blindness.
2. Argus II implant (FDA approved)


VPU
Camera
Glasses
External coil
B
Electrode cable
Array
Scleral band
C
Electrodes
Handle

Restores central vision Fig: 2

3.Vorti gene: gene therapy

"Poor prognosis"

II.Stargardt's disease 00:14:04

- Autosomal recessive
- Most common juvenile macular dystrophy
- Most common in children
- Decreases day vision, decreases central vision
- Etiology: mutation in ABC A_4 gene → code for protein which is needed for photo transduction

Mutation leads to → loss of protein

↓

- Abnormal accumulation of fatty material, which is toxic to macula.
- Lipofuscin accumulation also seen at macula.

- Clinical features: white flecks scattered throughout retina

"Pisiform in shape"

- Diagnosis:

  FFA: Dark choroid/silent choroid

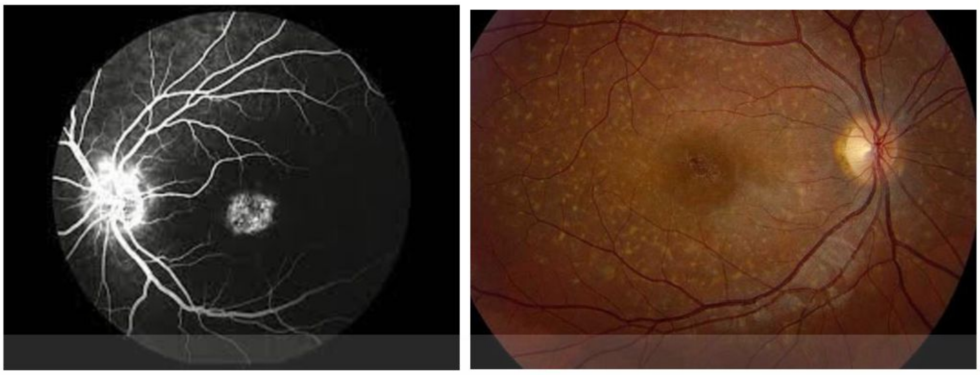


Fig: 3

III. Fundus Flavimaculatus 00:18:25

- It is a part of spectrum of stargard`s disease.
- Occurs in adults, A/K/A adult stargard's disease
- Better prognosis

IV. Best vitelliform dystrophy 00:19:08

- Autosomal dominant inheritance
- Occur as juvenile macular dystrophy
- C/O Day blindness

  Mutation of VMD_2 gene → Berforphin protein

  ↓

  Not synthesized

  ↓

  Accumulatio of lipofuschin at macula.

4 Stages

- Stage 1: previtelliform: Vision – normal

  EOG abnormal

TUTORIALS LOGIN FOR SUCCESS

Pigmentary changes ⊕

- Stage 2: vitelliform: Classic egg yok lesion

  Vision – mild decreases

- Stage 3 (pseudohypopyon): At puberty (lipofuchin starts going down absorbed) or layering of lipofuschin.
- Stage 4: Vitelleruptive

  ↓

  Breakup of material gives

  Scrambled egg appearance

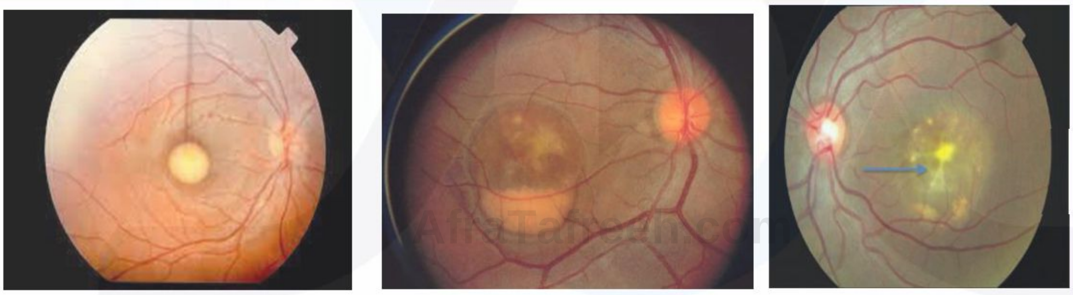


Fig: 4

- Normally EOG is 1.85

  <1.5 is best disease

TUTORIALS LOGIN FOR SUCCESS

Congenital Stationary Night Blindness 00:24:02

- It is group of disorders in which infantile nyctalopia is seen.

Two types

Normal retina

ERG Abnormal

↓

Oguchi's disease

Abnormal retina

ERG may be normal

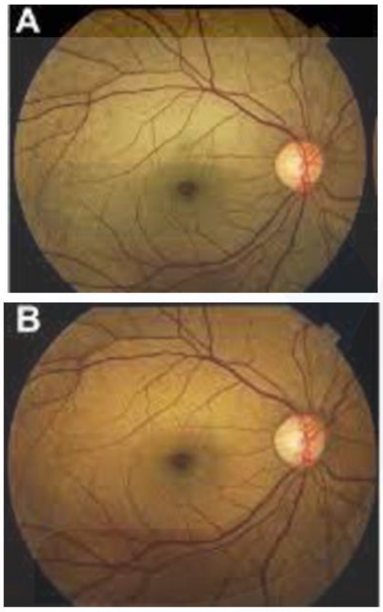


Fig: 5

Mizuophenomenon

Abnormal yellowish

&mge to retina ∵ rods

not functioning

↓

Dark adaptation to for

4-6 hours ∵ sitting in

dark room (eye open)

↓

Retina normal due to

refunctioning of rods

V. Leber congenital amaurosis 00:27:42

- Most severe retinal dystrophy causing blindness by 10yrs of age.
- No photo transduction
- Child has nystagmus
- Sluggish pupillary responses
- Decreased vision
- Association of cataract, keratoconus, epilepsy, mental retardation.

Occulodigital reflex present (repeated rubbing of eyes)

↓

- Keratoconus
- Fat atrophy
- Sunken eye.

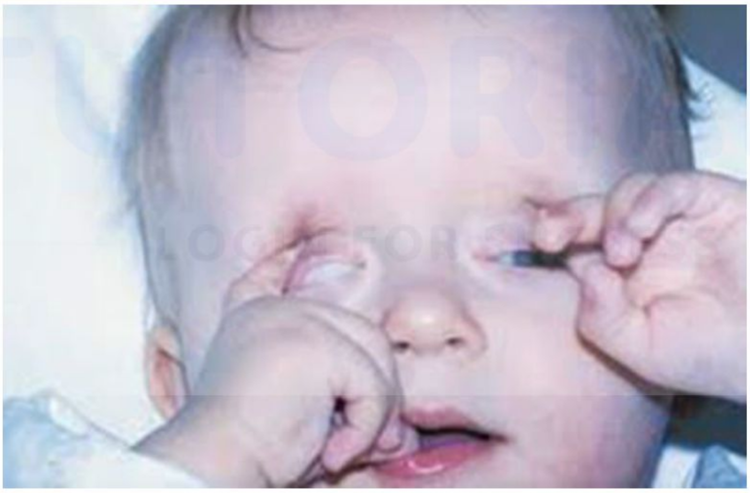


Fig: 6

RETINOPATHY OF PREMATURITY

- Normal process of angiogenesis in retina.

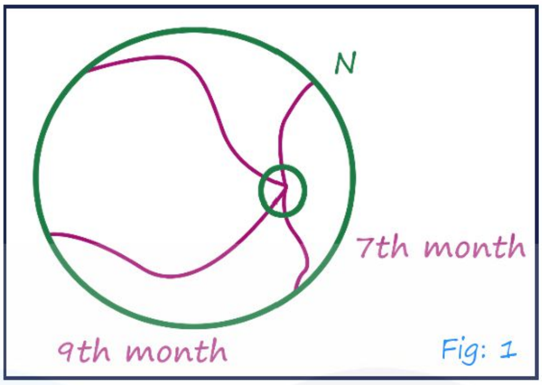


Fig: 1

- If the baby is born in 7th month

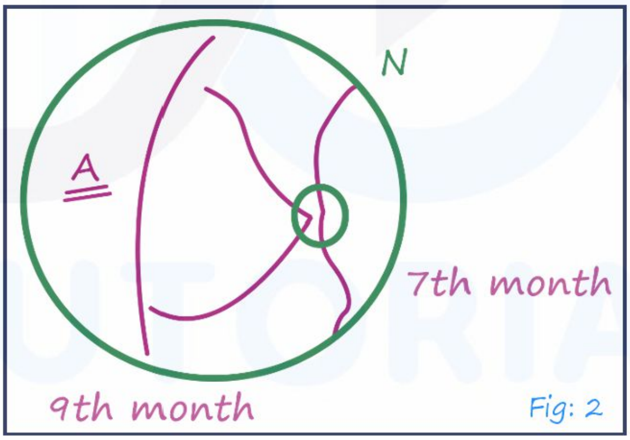


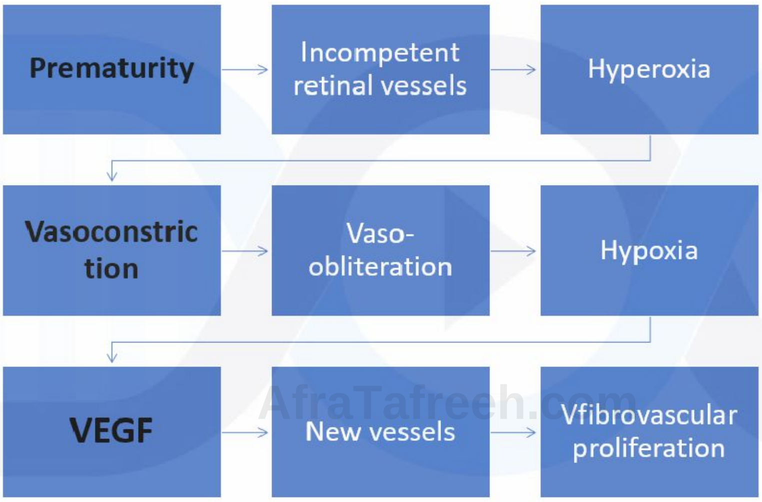
Fig: 2

Risk Factors 00:01:38

- H/O preterm birth → <34 weeks of gestation
- H/O low birth weight → < 2kg
- Even if baby is born after 34 weeks of gestation and has H/O. NICU admission may be due to hypoxia at birth or respiratory distress etc. where the baby is given oxygen supplement should be screened.

- If there are any of the risk factors mentioned above are seen then the baby should be screened for retina.

In womb After delivery
↓ ↓
Hypoxia → Hyperoxia → vasoconstriction
↓
Hypoxia


Prematurity
Incompetent retinal vessels
Hyperoxia
Vasoconstriction
Vaso-obliteration
Hypoxia
VEGF
New vessels
Vfibrovascular proliferation

- This leads to complication like tractional RD

International Classification of ROP (ICROP)

00:05:15

- ROP is categorized based on the severity of the disease into stages (0-5)
- Location of the disease (zone 1-3)
- Extent of disease based on clock hours (1-12)
- Presence of plus disease.

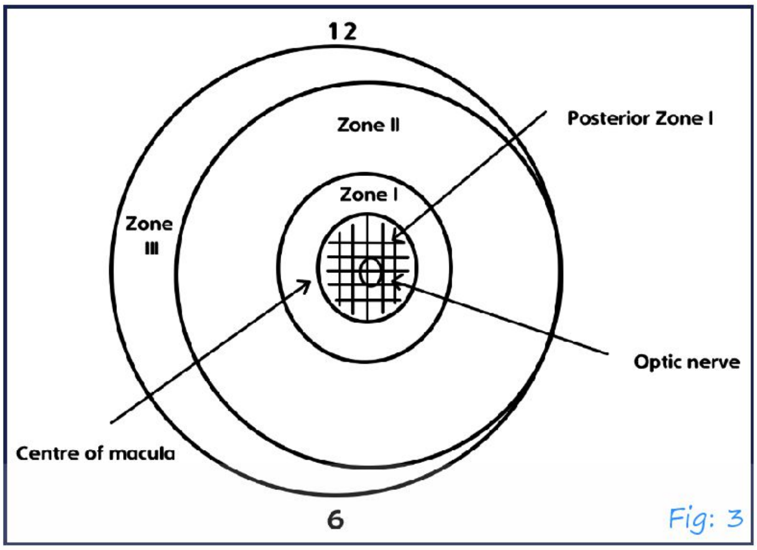

12
Zone II
Posterior Zone I
Zone I
Zone III
Optic nerve
Centre of macula
6

Fig: 3

- We take optic disk as center and zones are divided.
  - Zone I – distance b/w optic disc, fovea is doubled and taken as a radius for a circle this area is zone 1.
  - Zone II – circle – over nasal ora serata centered over zone I is zone II
  - Zone III – Remaining temporal crescent is zone III
- If ROP is more central – It is more dangerous

Disease extent

- Disease extent is recorded as clock hours 1-12 hours or as twelve 30° sectors or 360°
- The clock hours recorded is the total clock hours involved, not just the contiguous sectors.

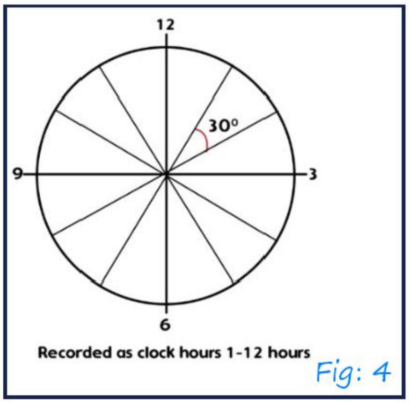

12
30°
9
3
6
Recorded as clock hours 1-12 hours

Fig: 4

Stages of ROP (1-5) 00:07:54

- Stage 1. Demarcation line
  - A thin but definite structure separating the avascular retina anteriorly from the posteriorly vascularized retina.

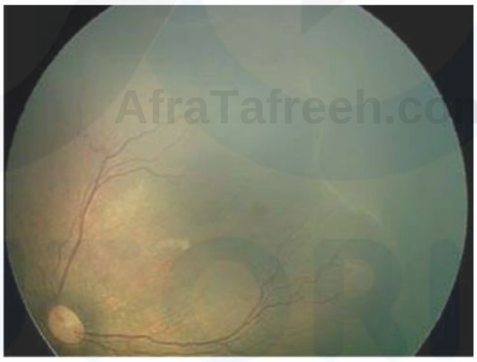


Fig: 5

- Stage 2 Ridge
  - A ridge arising from the demarcation line which has 3 dimensions (height and width) and extends above the retina.

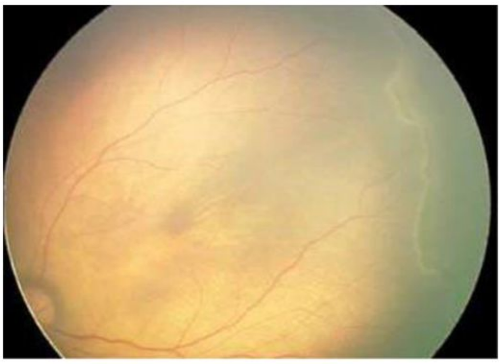


Fig: 6

- Stage 3 Extra retinal fibro vascular proliferation
  - Extra retinal fibrovascular proliferation or neovascularization extends into the vitreous from the ridge.

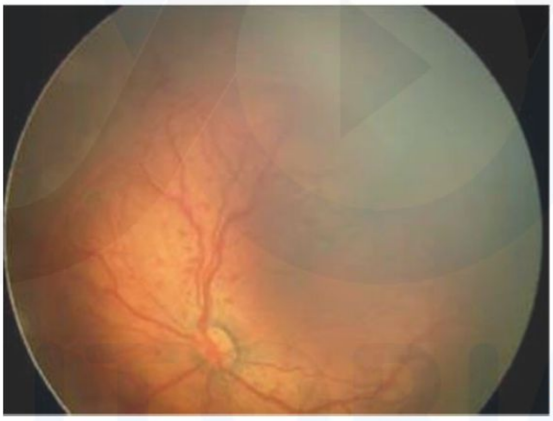
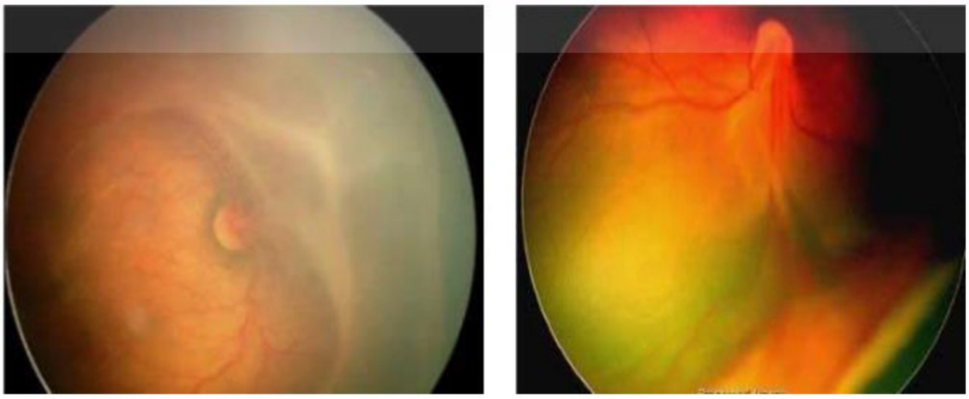


Fig: 7

- Stage 4 partial retinal detachment



4A: Extra foveal

4B: Foveal Fig: 8

- Stage 5. Total fibrovascular retinal detachment
  - Retinal detachments are generally tractional but may occasionally be exudative.
  - They are usually funnel-shaped.

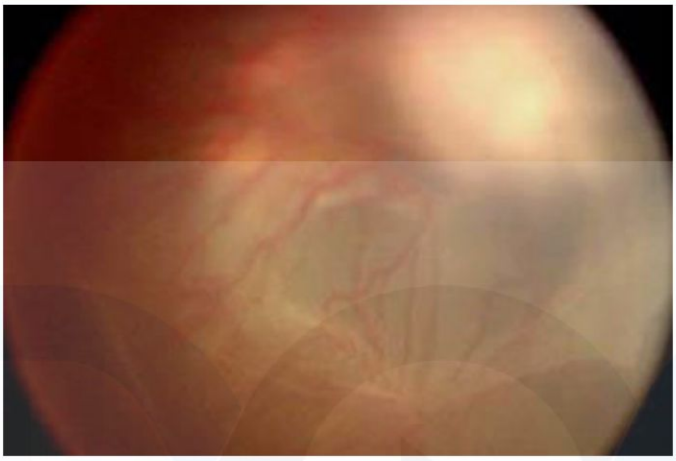


Fig: 9

ROP plus disease 00:09:05

- It is the presence of tortuous fully dilated vessel at posterior pole.
- Usually, it is in 2 quadrants
- There might be pupillary abnormality also

TUTORIALS LOGIN FOR SUCCESS

Screening 00:10:05

- If baby is <28 weeks of gestation
- If baby is <1200 gm

  These should screen early i.e., 2-3 weeks of birth
- If baby is born in between 28-34 weeks.
- If baby birth weight is 1200gm – 2 kg

  In these babies screening can be done even after 4 weeks of birth.

Treatment 00:11:30

- When to treat
  - If these is threshold ROP/ Type-1 – treatment is given threshold ROP → stage 3; zone 1 with plus sign for at least 5 continuous clock hours.

    (or)

    8 non continuous clock hours.
  - Type 1 ROP → problems in zone 1 at any stage with plus sign (or)

    Zone 1; stage 3 and without plus sign

    (or)

    Zone 2; stage 3/2, with plus sign
- How to treat
  - Laser photo coagulation – with diode laser

    For TRD → PPV is the only treatment
  - Breast feeding is preventive, stops further progression of ROP.

Aggressive ROP 00:14:08

- a.k.a aggressive posterior ROP
- Stage 1 $\xrightarrow{\text{quickly}}$ stage 5

MISCELLANEOUS TOPICS

Roth Spot 00:00:10

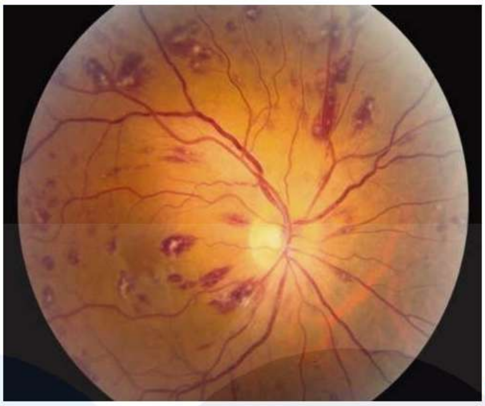


Fig: 1

- Flame shape hemorrhages with clear center.
- Classically they are seen in infective endocarditis
- They are also seen in
  - Diabetic retinopathy (rarely)
  - Dengue
  - Coagulation disorders.

TUTORIALS LOGIN FOR SUCCESS

Angioid Streaks 00:01:14

- Breaks/Dehiscence of bruch's membrane=

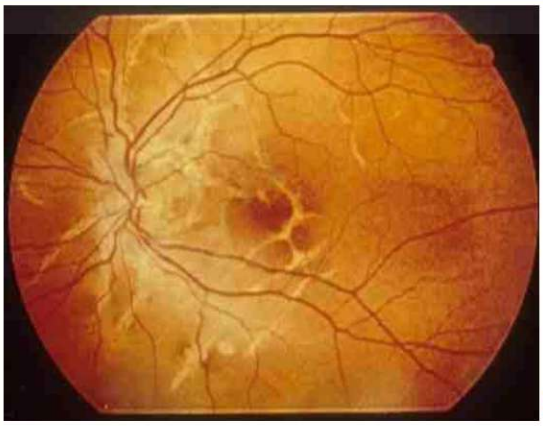


Fig: 2

- It gives the retina – a peau de orange appearance

Cause

- Mnemonic – PEPSI CAN cause DM
  - P – Pagets diseases
  - E – Ehler danlos syndrome
  - P – Pseudo Xanthoma elasticum
  - S – Sickle Cell Disease
  - I – Idiopathic
  - Hypercalcinosis
  - Acromegaly
  - Neurofibromatosis
  - DM
  - Myopia
  - Marfan syndrome

Bulls Eye Maculopathy 00:02:59

- It is seen as a zone of hyperpigmentation surrounded by hypopigmentation

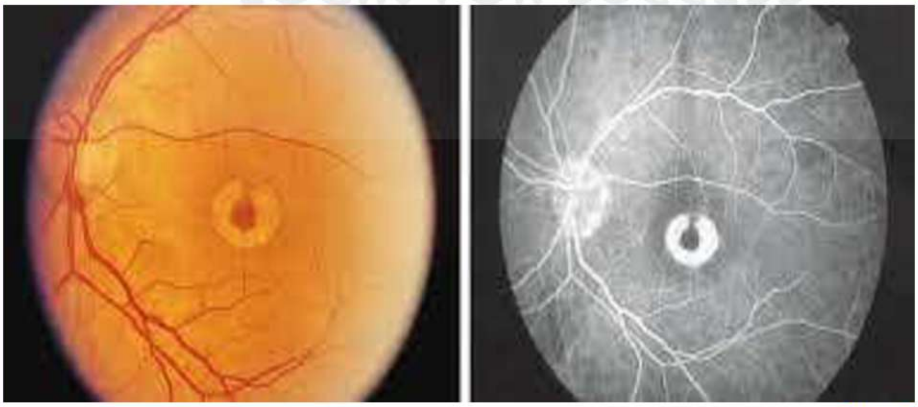


Fig: 3

- It is the problem in macula

- Cause – may be due to intake of certain drugs causing pigmentary changes at the level of macula.
- Cone Dystrophy
- Stargardt's disease
- Chloroquine retinopathy
- Clofazimine toxicity
- Age related macular degeneration
- Leber's congenital amaurosis
- Chronic macular hole
- Inverse retinitis pigmentosa
- It is the side effect of chronic use of chloroquine, HCQ

  i.e., HCQ > 5 mg/kg ⎤ for 5 years
  CQ > 2.3 mg/kg ⎦

Coats Disease 00:04:35

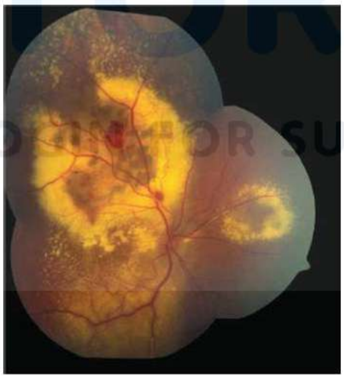


Fig: 4

- Idiopathic, telengectatic vessel with sub retinal or intra retinal exudation

Juxta Foveal Telengectesia (JFT) 00:06:21

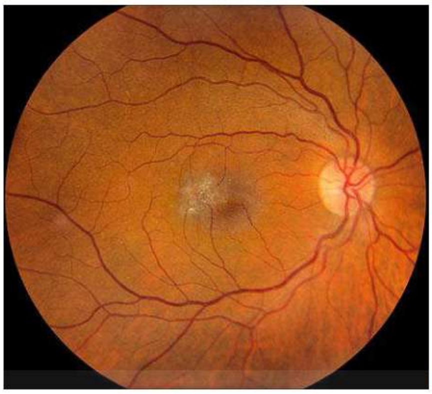


Fig: 5

- Classified into 3 types

  Type 1 – variant of coat's disease in adults

BASICS OPTIC NERVE

Optic nerve 00:00:34

- Axons of ganglion cells
- Approximately 1.2 million ganglion cells in each optic nerve
- Does not have neurilemma
- Cannot be regenerated
- Meningeal coats ⊕
- Myelin sheath ⊕ by oligodendrites

Optic disc / ONH – intraocular part

↓↓

Not myelinated

- If the optic disc is myelinated → MNF → Myelinated nerve fibres

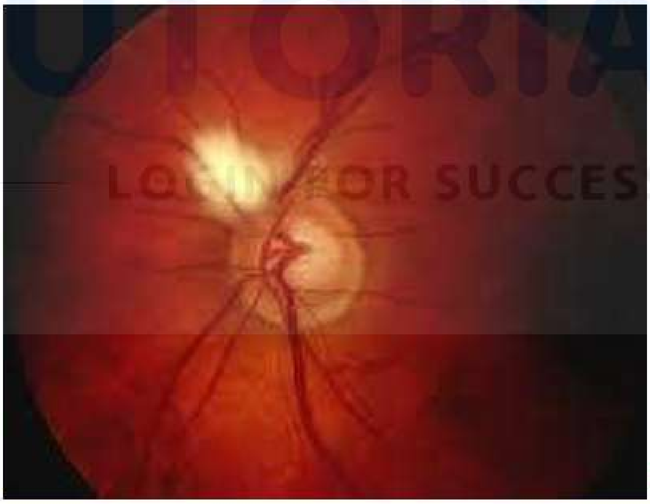


Fig: 1

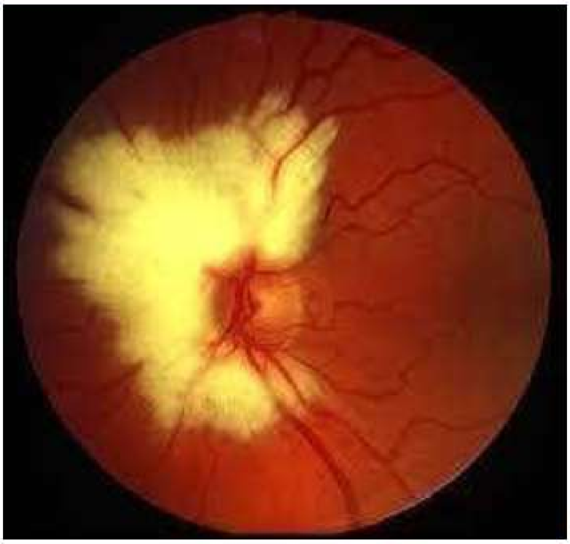


Fig: 2

- Myelination is associated with NF-1, strabismus

Parts of Optic Nerve 00:04:49

1. Intraocular part – Optic disc/optic nerve head
2. Intra orbital part – with orbit → longest portion – 30mm in length
   - In relation to CRA
     CRA enters 10 mm inferomedial to nerve

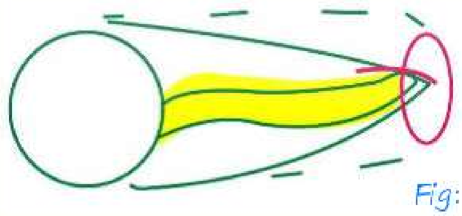


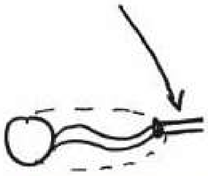
Fig: 3

- Optic nerve has a sinus course in the eye, it allows movement of eye.
- During blunt trauma, optic nerve not much injured because of its optic course

- Near annulus of zin, there is attachment of sheath of SR, MR

  -Whenever there is retrobulbar neuritis, → there is painful ocular movements

3.Intra canalicular part → Optic foramen → Optic canal



- Optic foramen → Optic canal
- In relation to ophthalmic artery
- Most likely to get injured by trauma

4.Intracranial part → ends at chiasma

- In relation to frontal lobe, cavernous sinus

Example: If you have frontal lobe, tumor, it will compress optic nerve on same side → I/L atrophic

- Increase ICT → meningeal compression → Bilateral papilledema

↓

Clinical papilledema because I/L atrophy

↓↓

Foster Kennedy syndrome

Functions of optic nerve 00:12:39

1.Vision → central and field of vision

→ best to check field is by perimetry

2.Color vision

3.Color contrast → Checked by Pelli Robson chart

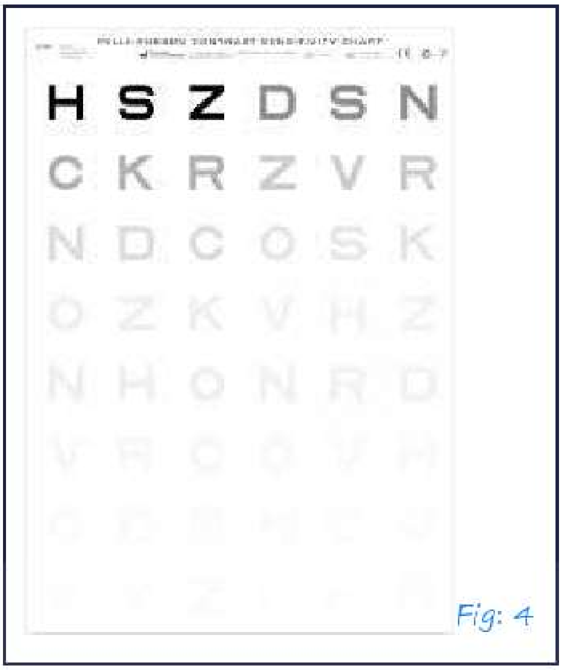


Fig: 4

Read at distance of 3 meters

4.Pupillary reaction – afferent – optic nerve

Congenital anomalies of ON 00:16:12

1.**Myelinated Nerve fibers**

- Myelin sheath over optic nerve head
- Slight astigmatism

2.**Morning glory syndrome** – mesodermal defect

- Lamina cribrosa absent
- Differentiation of arteries and nerve is gone

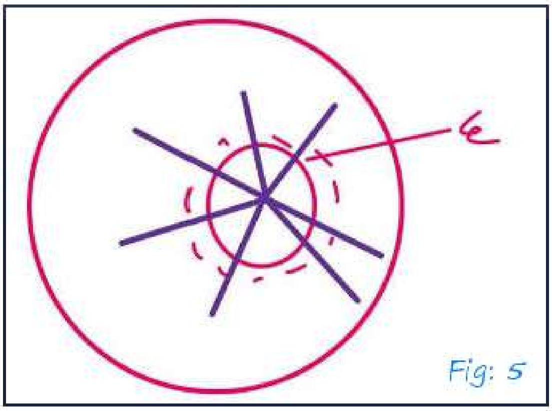


Fig: 5

- Spoke of wheel appearance

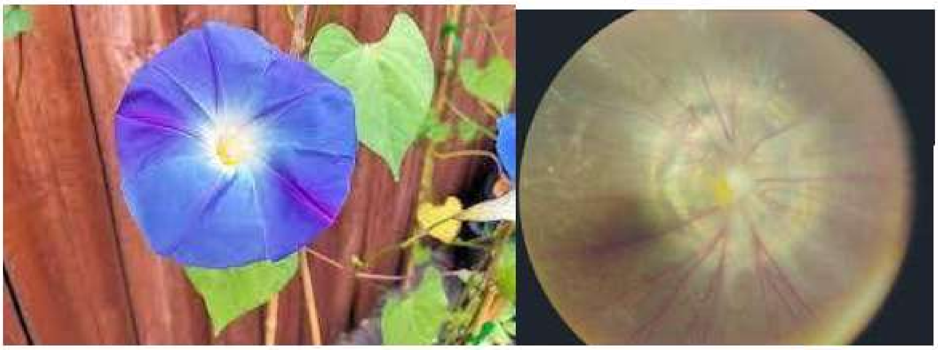


Fig: 6

3.Optic disc coloboma

- Coloboma – defect in closure of embryonic fissure
- Failure of closure of optic disc / hyaic fissure
- Excoriation in disc

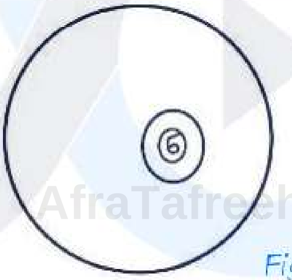


Fig: 7

TUTORIALS
LOGIC FOR SUCCESS

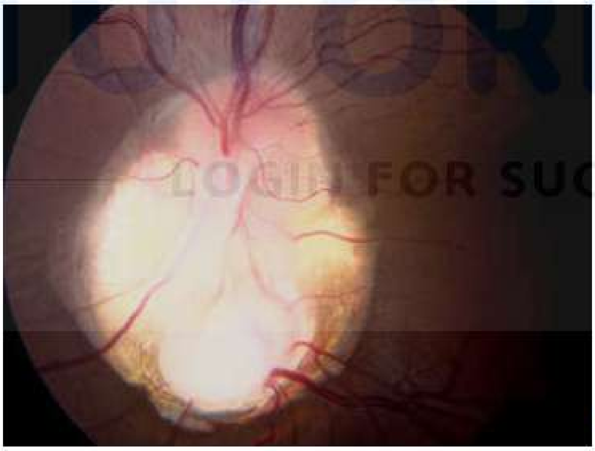


Fig: 8

4.Disc drusen

↓

Hyaline calcific material identified over disc

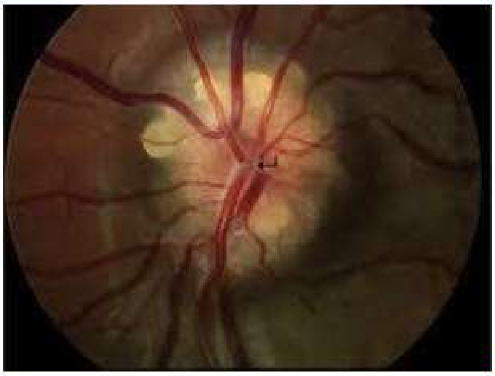


Fig: 9

- Autofluorescence → Disc drusen

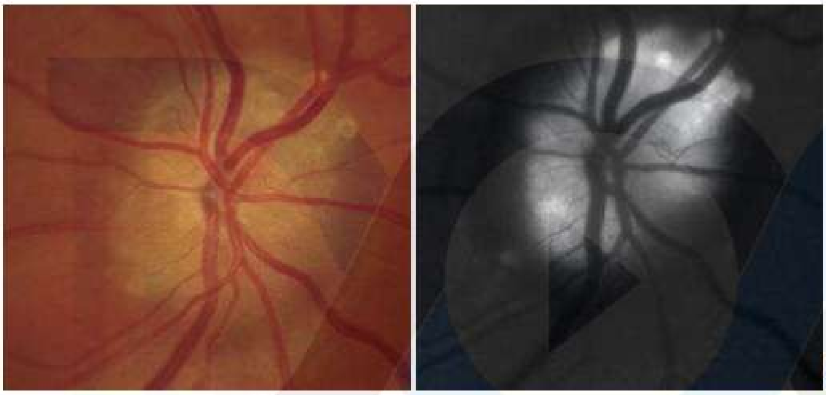


Fig: 10

TUTORIALS LOGIN FOR SUCCESS

5. Optic disc hypoplasia

- Decrease ganglion cells
- De monsier syndrome → absence of septum pellucidum
- Agenesis of corpus callosum

Hypoplasia of optic nerve

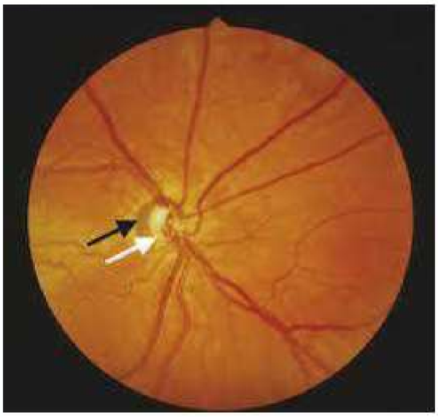


Fig: 11

A. Hypoplasia of the optic nerve

B. Hypoplasia of the right and left optic nerves

C. Hypoplastic pituitary stalk

D. Complete absence of the septum pellucidum

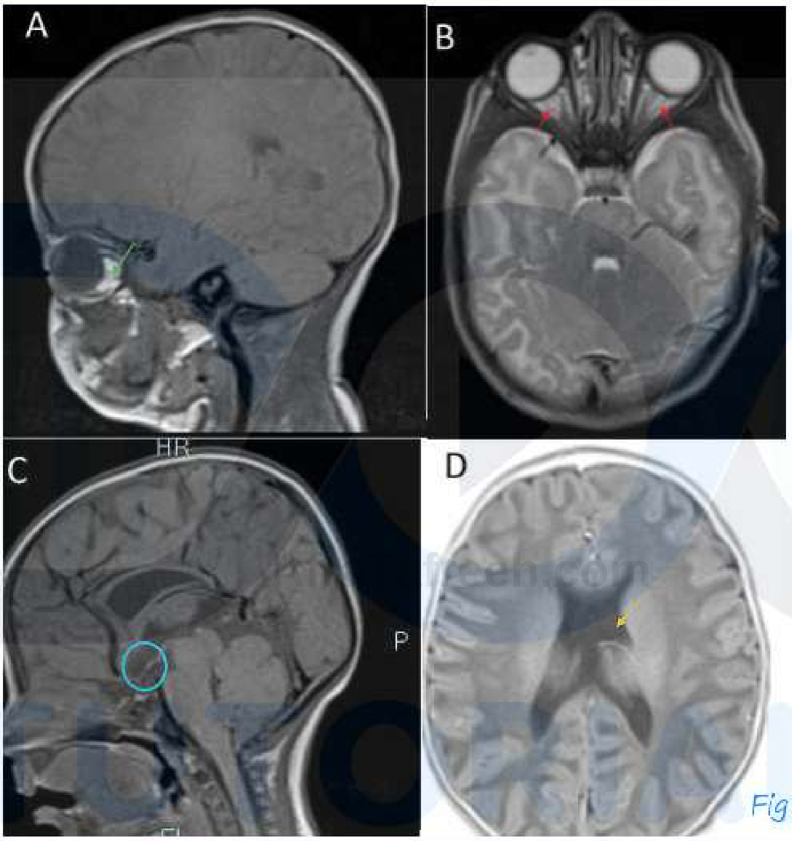


Fig: 12

- Double ring sign

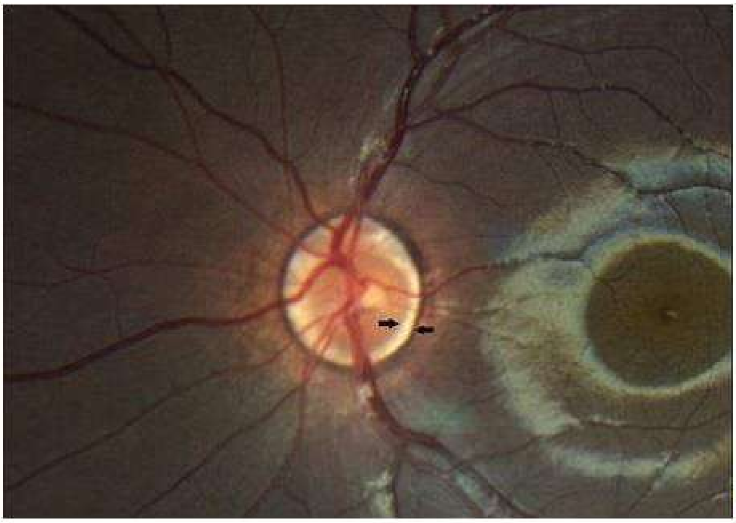


Fig: 13

The outer ring – junction between the sclera and the lamina cribrosa

Inner ring abnormal extension of RPE with the outer portion of the lamina cribrosa

6.Optic disc pit

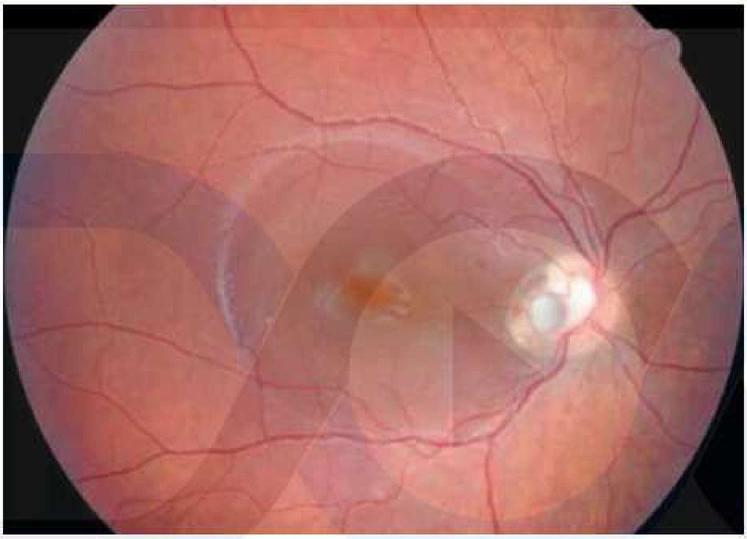


Fig: 14

- Greyish pale region usually on temporal side of disc
- Most common temporal

Associated with NSD / serous detachment at the level of macula

Blood supply of optic nerve head

Blood supply of optic nerve 00:23:31

Blood supply of optic nerve head

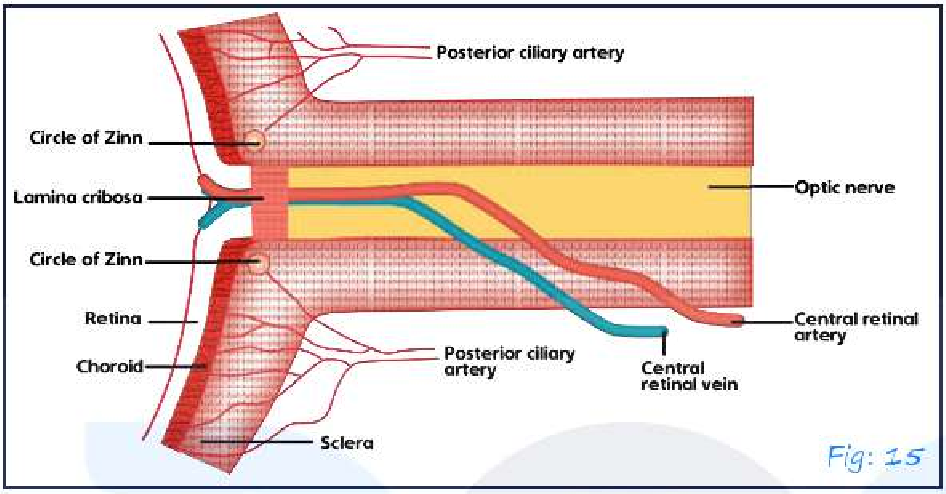

Posterior ciliary artery
Circle of Zinn
Lamina cribosa
Circle of Zinn
Retina
Choroid
Sclera
Optic nerve
Central retinal artery
Posterior ciliary artery
Central retinal vein

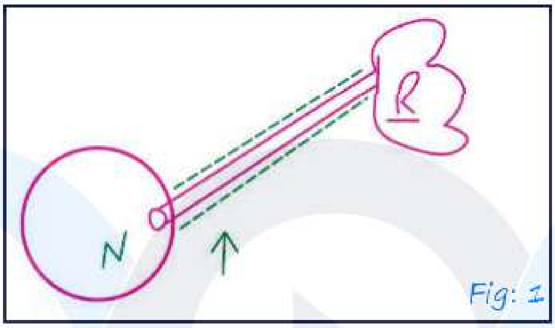
Fig: 15

- Surface layer – retinal arterioles
- Prelaminar region- centripetal branches of peripapillary choroid and vessels of lamina cribrosa
- Lamina cribrosa – PCA and arterial circle of Zinn
- Retrolaminar part – branches from CRA and pial plexus.

PAPILLEDEMA AND OPTIC NEURITIS

Papilledema 00:00:34

- Papilla means disc
- Oedema means swelling
- Reactionary disc oedema because of increase ICT



Axon around lamina cribrosa

↓

Stasis

↓

Extracellular edema venous congestion

Increased intracranial pressure with increased CSF pressure

↓

Increased pressure transmitted along subarachnoid space to optic nerve

↓

Axoplasm flow blocked

↓

Stasis in optic nerve head

Extracellular edema (Swelling of axonal fibres)	Venous congestion (Secondary vascular change)

Symptoms

- Headache severe throbbing
- Nausea
- Projectile vomitings
- Transient blurring of vision – amaurosis fugax

↓

Because of transient ischemia of nerve

Signs

- Pupil → normal
- Retina

1.Blurring of nasal margin → 1st sign

2.Grossly enlarged disc

3.Venous congestion, tortuous dilated

4.Hemorrhage / exudate

SVP

- Spontaneous venous pulsation → normally seen in 80%, absent in 20%
- Absent SVP – sings of papilledema earliest
  - Angry sun appearance

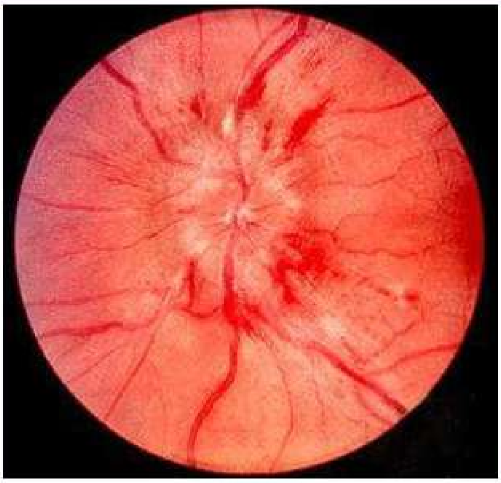
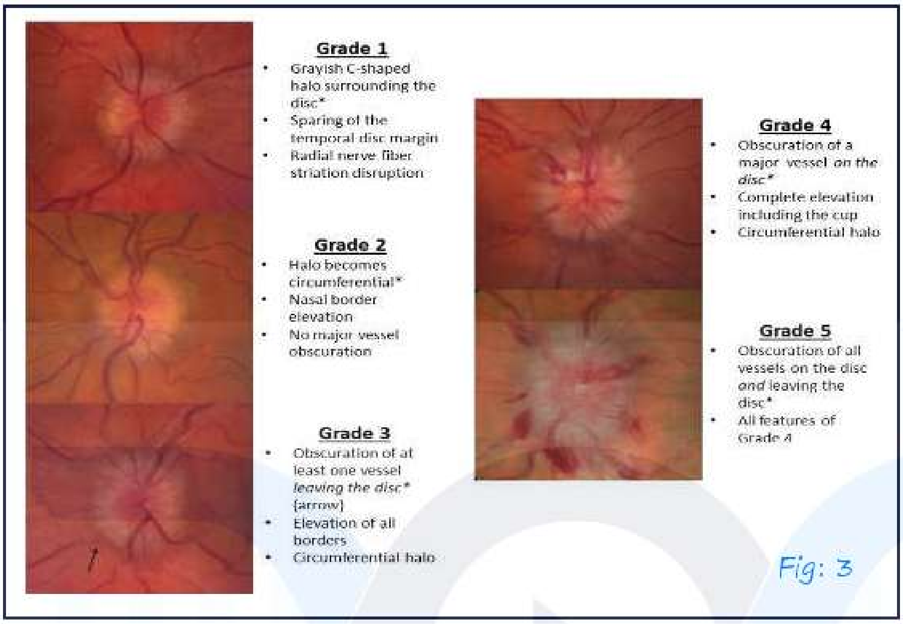


Fig: 2

- Always bilateral
  - Frisen grading



Grade 1

- Grayish C-shaped halo surrounding the disc*
- Sparing of the temporal disc margin
- Radial nerve fiber striation disruption

Grade 2

- Halo becomes circumferential*
- Nasal border elevation
- No major vessel obscuration

Grade 3

- Obscuration of at least one vessel *leaving the disc** (arrow)
- Elevation of all borders
- Circumferential halo

Grade 4

- Obscuration of a major vessel *on the disc**
- Complete elevation including the cup
- Circumferential halo

Grade 5

- Obscuration of all vessels on the disc *and* leaving the disc*
- All features of Grade 4

Fig: 3

- Visual field → no scotoma

Optic nerve head corresponds to blind spot

↓ ↓↓

↑↑ enlarged ⟶ enlarged

- Blind spot

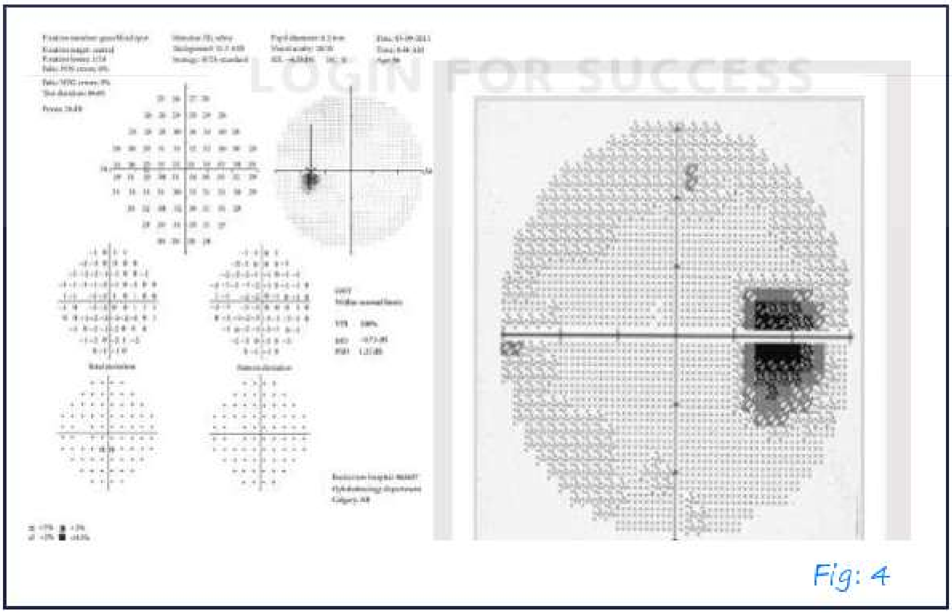


Fig: 4

Causes of enlarged blind spot

1. Papilledema
2. Myopia
3. Megalopapilla
4. Drusen

- Chronic papilledema

i. Champagne cork appearance → Vintage papilledema

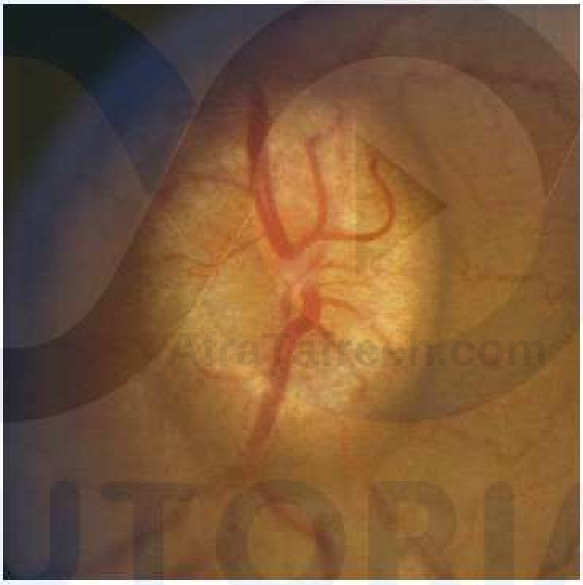


Fig: 5

ii. Optocillary shunt → abnormal shunk vessels

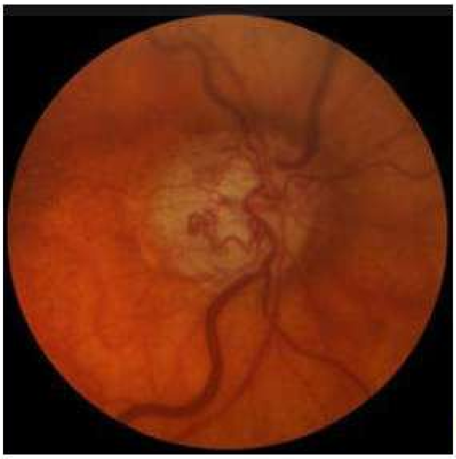


Fig: 6

iii. Corpora amylacea – accumulation of amyloid material

iv. 2° optic atrophy → vision lost and abnormal pupil

Paton line

↓

Surrounding retina thrown into fold

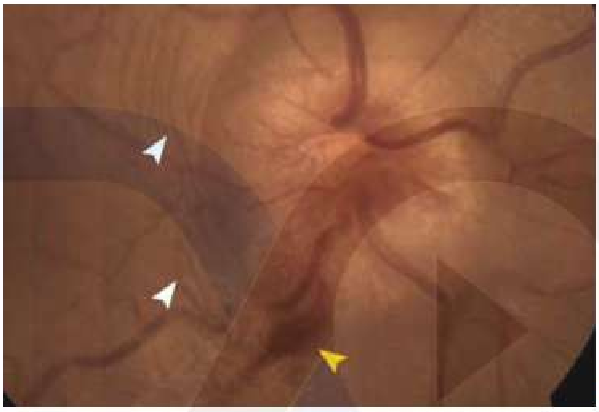


Fig: 7

Idiopathic intracranial HTN 00:15:03

- Also called BIH / Pseudotumor cerebri
- Increased ICT, papilledema without and primary tumour or mass lesion
- Young obese female → headache and tinnitus
- Causes
  - N – Nalidixic acid
  - O – Obesity
  - N – Nitrofurantoin
  - S – Steroids
  - T – Tetracycline
  - O – OCP
  - P – Phenothiazine

Modified dandy critic

Modified dandy criteria
Symptoms of increased intracranial pressure (i.e. nausea, vomiting, papilledema, or transient visual disturbances)
No focal neurological signs with the exception of abducens nerve palsy, unilateral or bilateral
Elevated ICP (CSF opening pressure >25cm H_2O) on a lumbar puncture with an unremarkable CSF composition
CT imaging without an etiology for the increased ICP (i.e. obstructive hydrocephalus, mass lesion, structural abnormality, and / or vascular lesions)

Optic neuritis 00:18:01

TUTORIALS LOGIN FOR SUCCESS

- Optic nerve inflammation
- 3 types based on location of Optic nerve inflamed

1.Papillitis

- Intraocular part
- Most common type children infection s
- Clinical features → sudden loss of U/L vision
- Pupil – RAPD
- Retinal – disc swelling < 2 times

2.Retrobulbar neuritis

- Inflammation of intra orbital nerve part
- Most common type in adults

- Most common cause – MS (Deric disease)
- Clinical feature: -
  - Sudden U/L loss of vision
  - Painful ocular movements
- Signs: -
  - Retina normal
  - Pupil – abnormal RAPD
- MRI – to confirm diagnosis

3. Neuroretinitis → Papillitis + CME
   - RNFL Edema → macular star
   - Post viral / vaccinations

- 2 phenomenon – optic neuritis / RBN
  - i. Pulferick's phenomenon: - Altered perception of moving objects
  - ii. Uthoff's phenomenon: - Worsening of symptoms to heat
- Management
  - Optic neuritis treatment trial

    ↓

    I/V methyl prednisolone 1d/day X 3 days

    ↓

    Oral steroids X 11 days
  - If treated with oral steroids alone → there is chances of recurrence
    - Papillitis

TUTORIALS LOGIN FOR SUCCESS

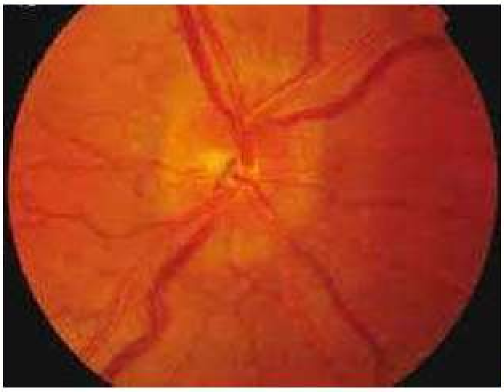


Fig: 8

- Neuroretinititis → macular star appearance

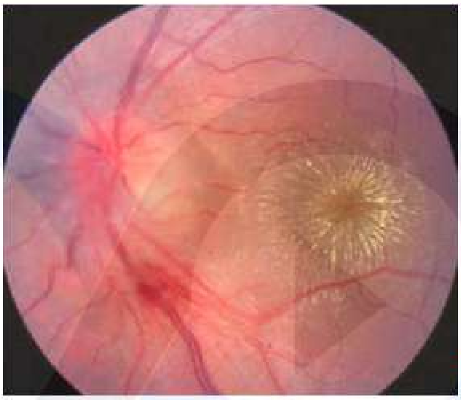
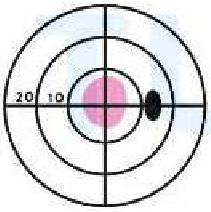
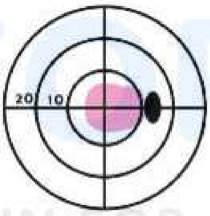


Fig: 9

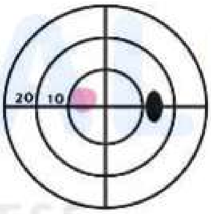
- Perimetry findings



A central scotoma involves the point of central fixation. It's always associated with decreased visual activity



A central scotoma involves the point of central fixation and the area between the blind spot and the fixation point



A paracentral scotoma affects an area of the visual field that is nasal or temporal to the point of central fixation

OPTIC ATROPHY

- Def: Death of ganglion cells
- Divided into various types based on etiology and clinical picture

Types 00:00:33

Primary

- T – Tumor
- T – Trauma
- T – Toxin
- N – Nutritional deficiency → Vit B1, B6, B12 → Characteristic
- H – Hereditary

Toxins

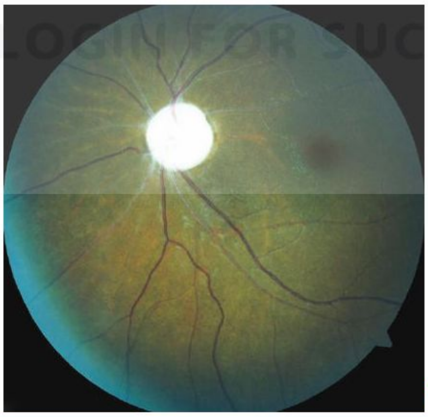


Fig: 1

- Pneumonic – Medical
- M – Methanol
- E – Ethambutol

- D – Digoxin
- I – INH
- C – Cyanide (Tobacco) chloramphenicol
- A – Aspirin, Amiodarone
- L – Lead
- Primary optic atrophy → Chalky white optic disc
- Pale chalky white disc
- Margins are sharp

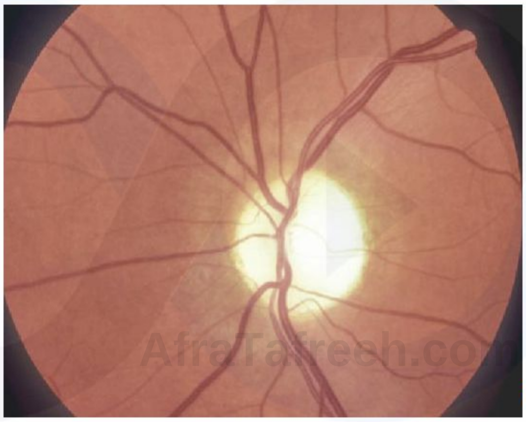


Fig: 2

Hereditary

- 1.Leber hereditary optic neuropathy (LHON)
- Point mutation at 11778 location
- Mitochondrial inheritance

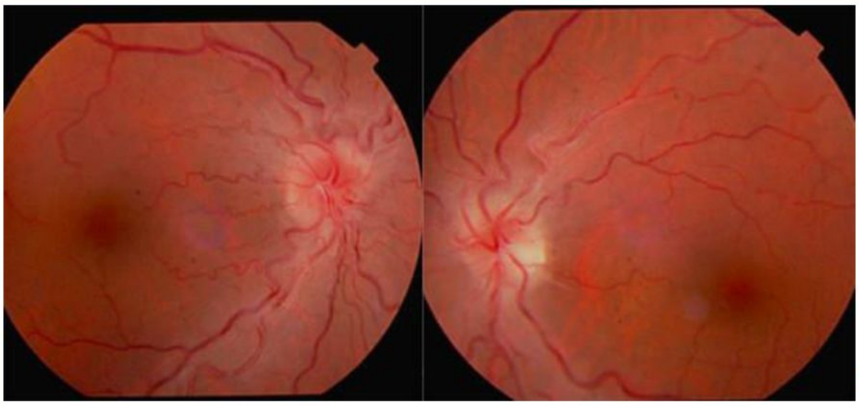
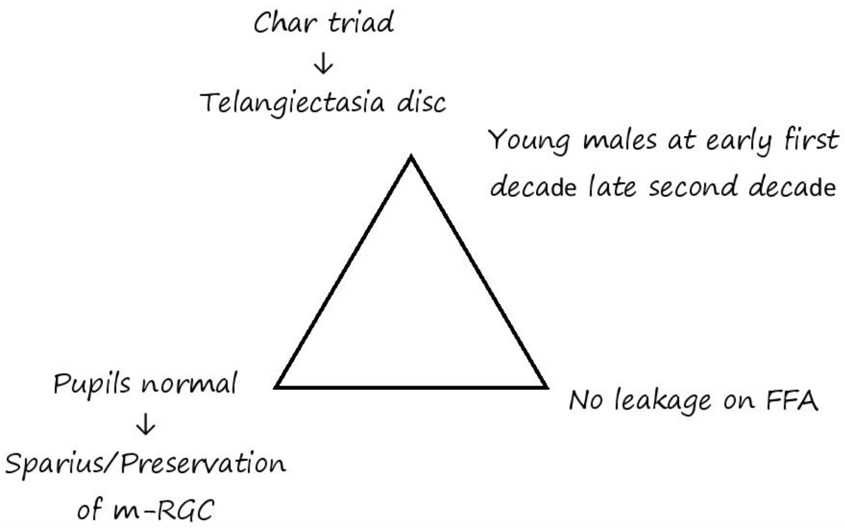


Fig: 3

TUTORIALS
LOGIC FOR SUCCESS


Char triad
↓
Telangiectasia disc
Young males at early first decade late second decade
Pupils normal
↓
Sparius/Preservation of m-RGC
No leakage on FFA

- 2.Kjer – Autosomal dominant
- Sensory neural deafness
- 3.Behr - autosomal recessive
- 4. Wolfram/ DIDMOAD

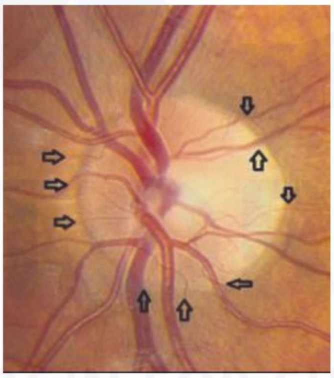


Fig: 4

DOC TUTORIALS LOGIN FOR SUCCESS

Kestebaum index 00:07:15

- Small normal vessel are found on edge of disc
- Normal disc → Ratio of small vessels is generally >10
- In optic atrophy the ratio becomes <10

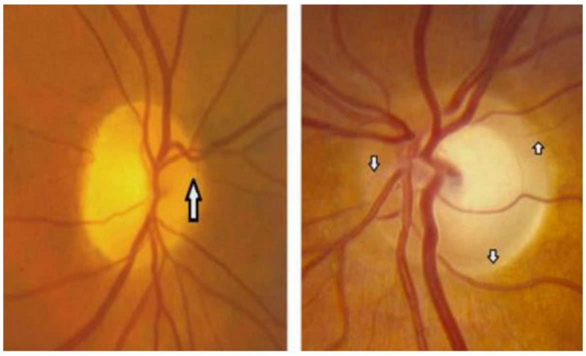


Fig: 5

Secondary

- Post inflammatory/Prior swelling of disc
- Papilledema
- Papillitis
- AION (Non arteritis)

Not treated on time
↓
secondary optic atrophy

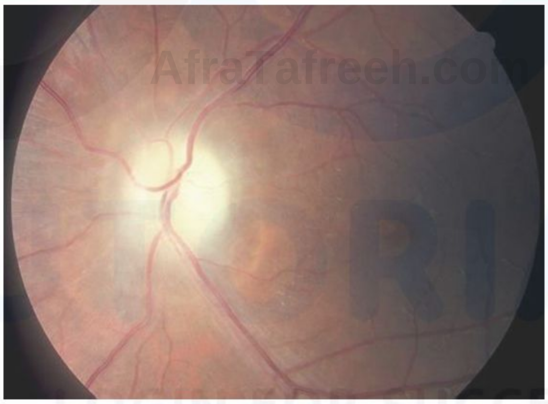


Fig: 6

- Grey white
- Gliosis
- Margins are not sharp

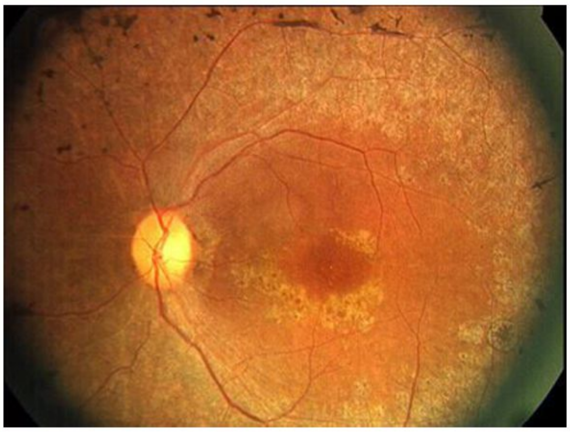


Fig: 7

- Retinitis pegmentosa → Bony spicules seen
- Degenerative changes

Consecutive

- Follows retinal degenerations like :
- Retinitis pigmentosa
- PRP
- CRAO
- Pathological myopia

Glaucomatous

- IOP
- Also called as cavernous optic atrophy

ANTERIOR ISCHEMIC OPTIC NEUROPATHY

- Anterior ischemic optic neuropathy
- Infarction of posterior ciliary artery (PCA is a branch of choroidal artery)
- Depending upon etiology AION divided into 2 types

Arteritic

↓

Mainly giant cell arteritis (GIA)

Non arteritic

	Arteritic	Non arteritic
Causes	Giant Cell Arteritis	DM, HTN, Arterial sclerosis
Age group	>70years	20-70 years
Presentation	Sudden gross decreases in vision	Not so gross
Pain jaw claudication ct	(+)	(-)
Optic disc	Pallor disc edema	Hyperaemia of disc

Tenderness of temporal artertry	(+)	(-)
ESR	↑↑	(N)
Treatment	Steroids (Urgent)	Carbidopa + Levodopa
Prognosis	Poor	Good
	Ocular emergency because these is involvement of other eye also	

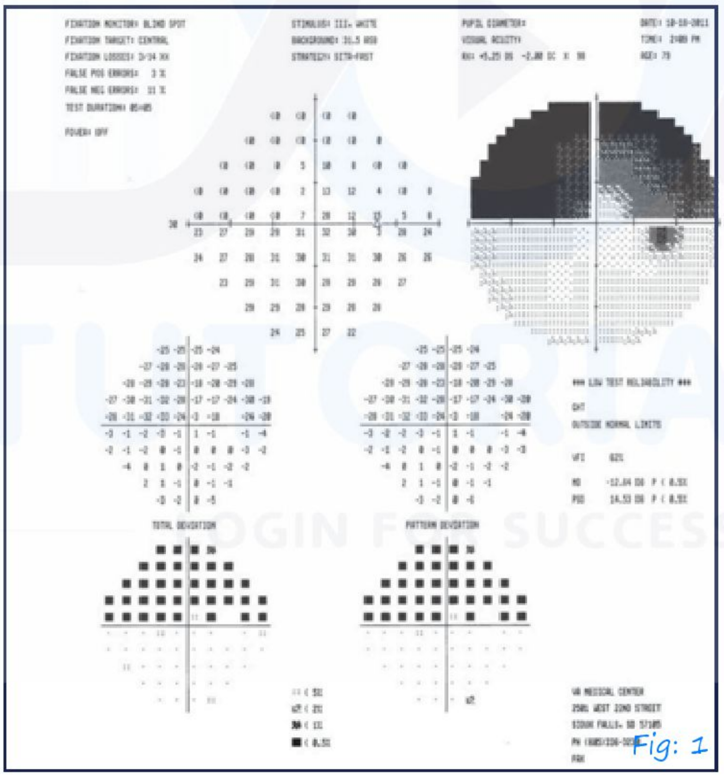


Fig: 1

Altitudinal field defect

Pseudo foster Kennedy syndrome 00:05:32

- Unilateral optic atrophy with C/L optic disc edema
- Eg in bilateral sequential AION
- NAION – More commonly seen
- PEKS – Can be noted in these patients as new acute visual loss anal optic disc swelling in one eye with prior visual loss and secondary optic disc atrophy resulting from a previous ischemic event in other eye

VISUAL FIELD PATHWAY

Visual Pathway and Defects

- Begins with rods and cones
  ↓
  1st order for visual pathway

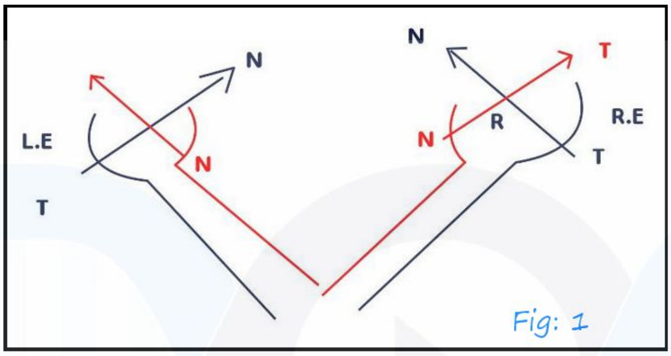


Fig: 1

- Rods & cones
  ↓
- Bipolar cells → 2nd order neurons
  ↓
- Ganglion cells → 3rd order neurons
  ↓
- Optic nerve
  ↓
- Optic chiasma
  ↓
- Optic tract
  - R → Right Temporal &
    → Left nasal fibers
  - L

  ↓
- Lateral geniculate body → 4th order
  ↓
- Optic radiations
  ↓

- Visual cortex
  - primary visual area – 17
  - Secondary visual area – 18,19

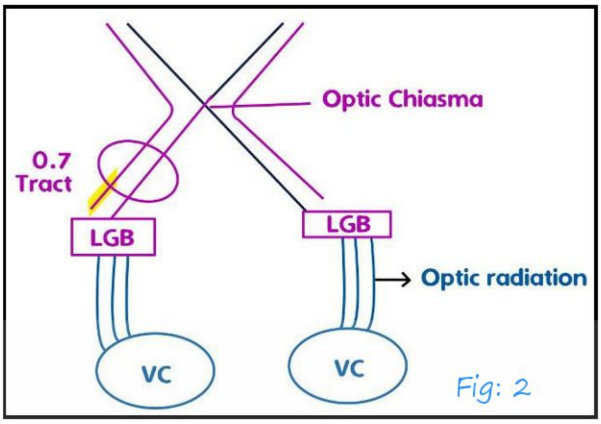

Optic Chiasma
0.7
Tract
LGB
LGB
Optic radiation
VC
VC

Fig: 2

- Anopia: – total loss of filed
- Hemianopia: – loss of ½ of flied
- Quadrantanopia: – loss of ¼ of field
- Homonymous hemianopia: – loss of ½ of filed is similar

Visual field defects 00:09:56

- Lesion @ L optic nerve

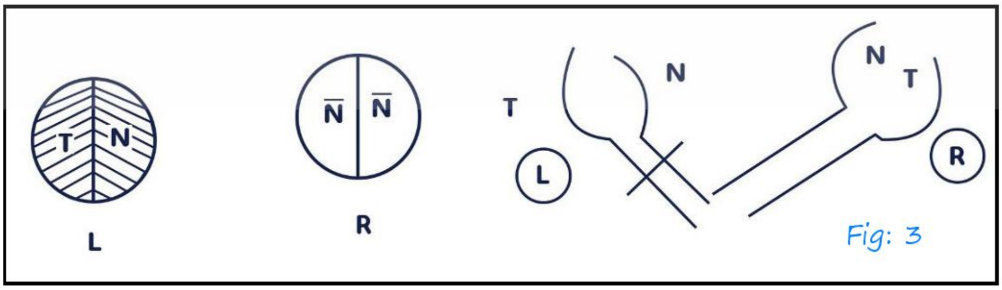

T
N
L
N̄
N̄
R
T
L
N
N
T
R

Fig: 3

- Left Temporal fibers and left nasal fibers defect

↓

Left anopia

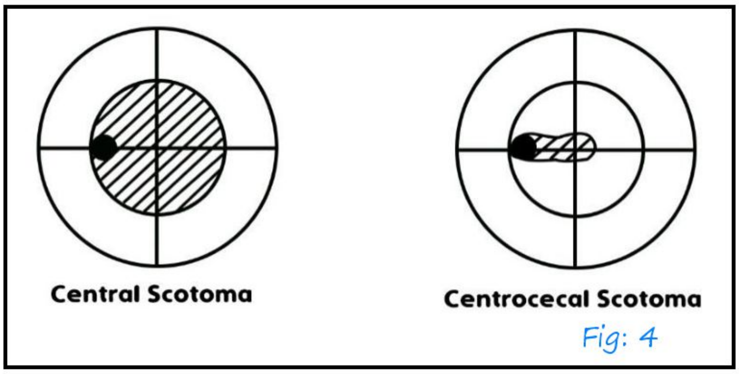

Central Scotoma
Centrocecal Scotoma

Fig: 4

- Central fixation point gone and extends to blind spot → Centro cecal scotoma → papulocular bundle, ischemic compression

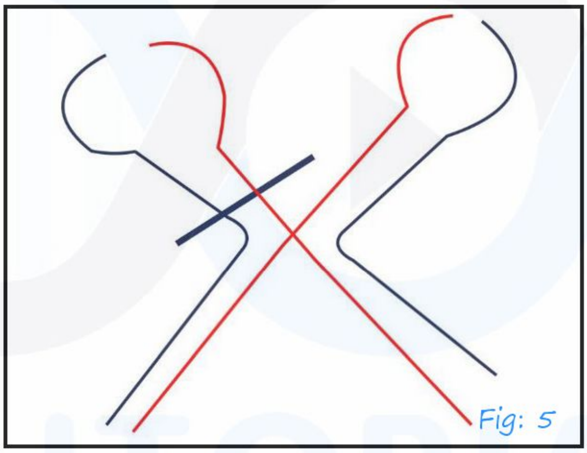


Fig: 5

Lesion at proximal part of Left optic nerve

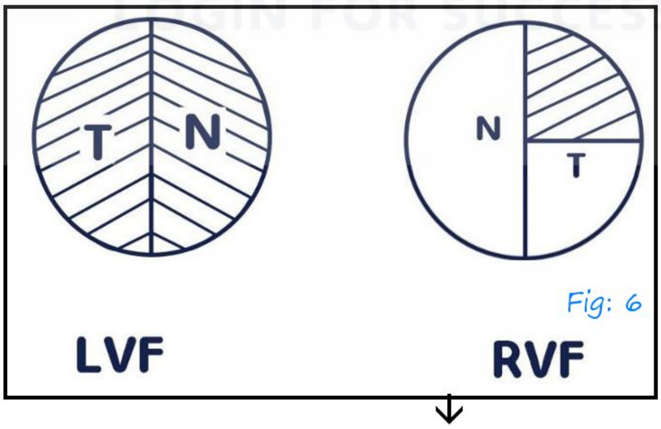

T
N
N
T
LVF
RVF

Fig: 6

↓

Junctional scotoma of Traquain errors

- Left Proximal nerve has inferonasal fibers from Right optic nerve → make a loop known as non-Willebrand knee

↓

Right supero temporal field loss

Lesion at chiasma

- Pituitary gland is present below chiasma

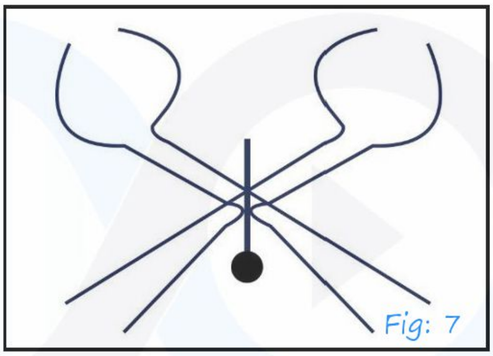


Fig: 7

- If pituitary gland tumor is enlarging → compression of central part of chiasma
- Central part → crossing /decussation of fibers

TUTORIALS LOGIN FOR SUCCESS

Lesion @ central chiasma

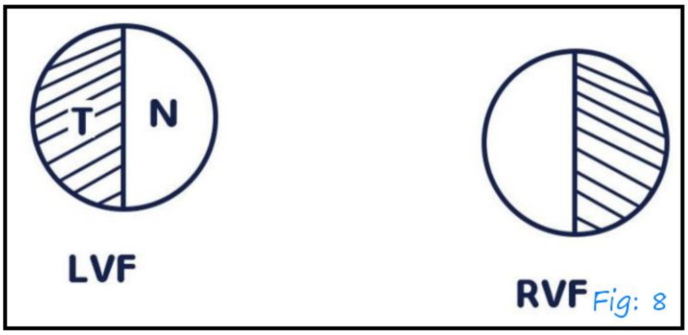


Fig: 8

↓

Heteronymous hemiopia/ Bitemporal

- Ex. seen in macroadenoma > 10 mm
  - craniopharyngiomas
  - 3rd ventricular glioma
  - Anterior cerebral aneurysm
- Q. Earliest filed defect in pituitary tumor – 1st affects inferonasal fibers of both side

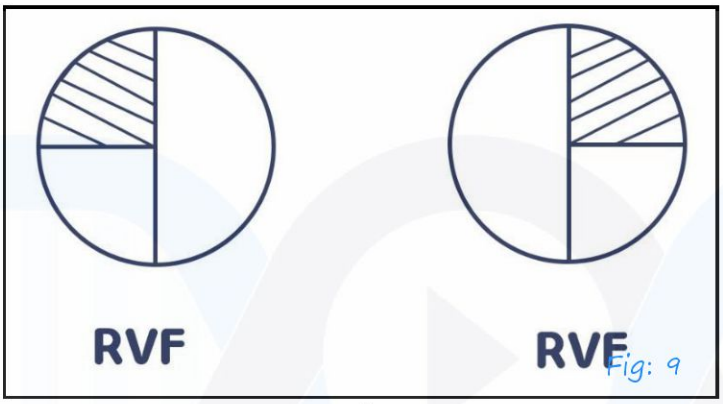


Fig: 9

↓

Bitemporal heteromyopia superior quadrantopia

- Q. Earliest VF defect in craniopharyngioma

↓

Affects supero nasal fibers

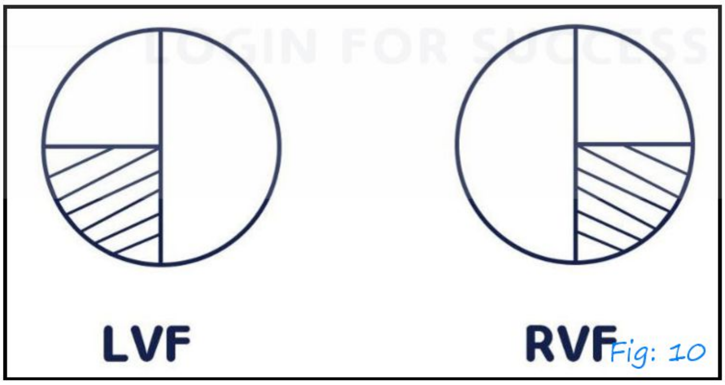


Fig: 10

↓↓

Bilateral heteronymous inferior quadrantopia

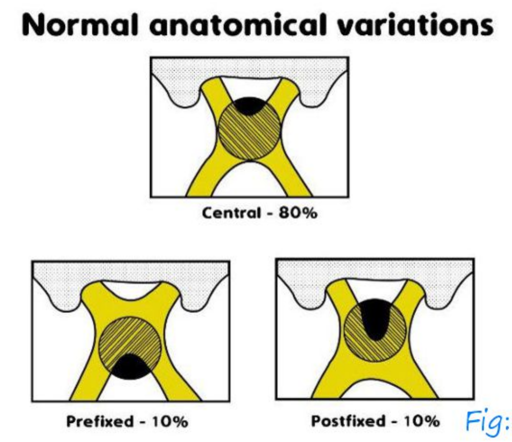

Normal anatomical variations
Central - 80%
Prefixed - 10%
Postfixed - 10%

Fig: 11

- Normal anatomic variation
- Prefixed → Cross over is anterior
- Postfixed → Cross over is posterior
- Central: lies directly over Sella, expanding pituitary tumour involves chiasma first.
- Pre-fixed: lies more anteriorly over tuberculum sellae, pituitary tumour involves optic tract first
- Post-fixed: lies more posterior over dorsum sellae, pituitary tumour damage optic nerve first

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TUTORIALS
LOGIN FOR SUCCESS

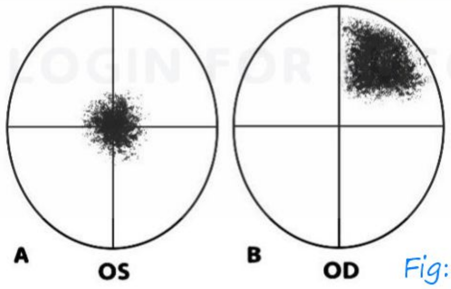


Fig: 12

Junctional scotoma

↓

Lesion on Left optic nerve proximal part

- OS- Ocular sinister
- OD- Oculars dextrus

Lesion at Left optic tract

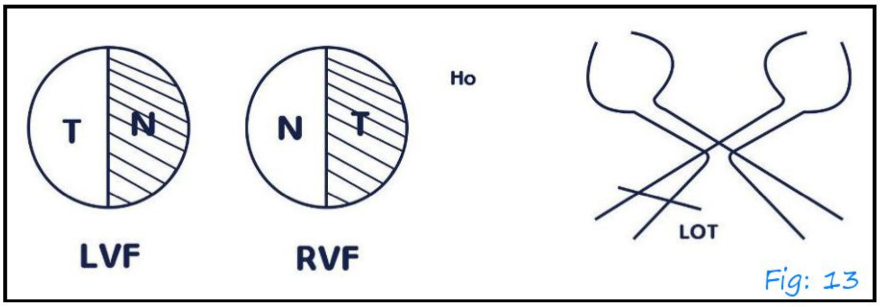


Fig: 13

- Left optic tract affects Left temporal part
  ↓
  Left nasal field gone
- Left optic tract affect Right nasal part
  ↓
  Right temporal field gone

Homonymous hemianopia

- Lesion at optic nerve → Right Homonymous hemianopia
  → Wernicke hemianopic pupil
  → Optic atrophy

TUTORIALS LOGIN FOR SUCCESS

Lesion @ Left lateral geniculate body

- The crossed fibers end in lamina 1, 4and 6
- Uncrossed fibers end in the lamina 2, 3and 5.
- The layers 1 and 2 - magnocellular
- Layers 3-6 - parvocellular.
- Magnocellular layers receive their visual input from the large Y ganglion cells of retina- movement and flicker.

- Parvocellular layers receive their input almost entirely from the X-ganglion cells - color vision, texture, shape and fine depth
- Right homonymous hemianopia
- Key hole defect → incomplete LGB lesion

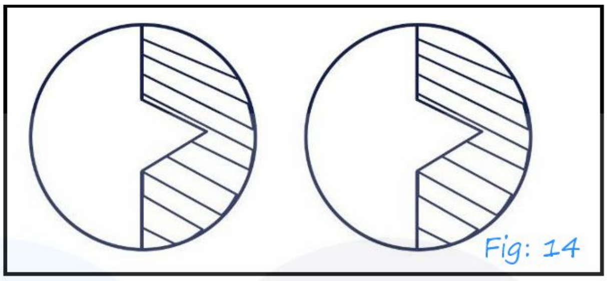


Fig: 14

- Pupil- normal
- Optic atrophy- N

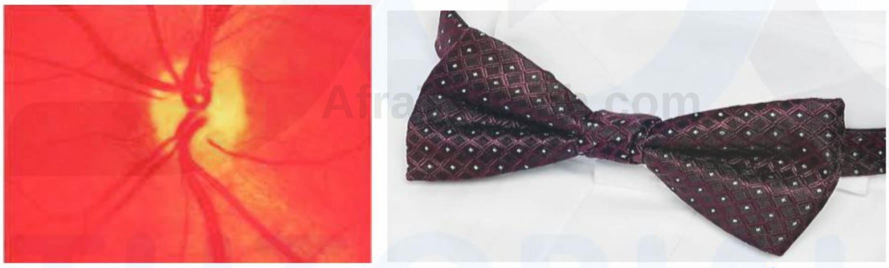


Fig: 15

- Bow tie OA → Superior and inferior filed intact
  → Nasal and temporal field affected

Lesion at Left optic radiations

- Right Homonymous hemianopia

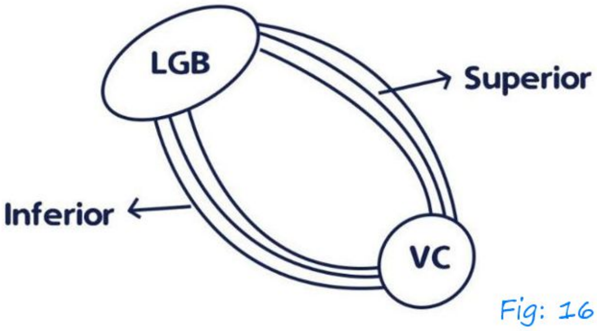


Fig: 16

- When fibers of optic radiation are going from LGB to VC, they make 2 loops

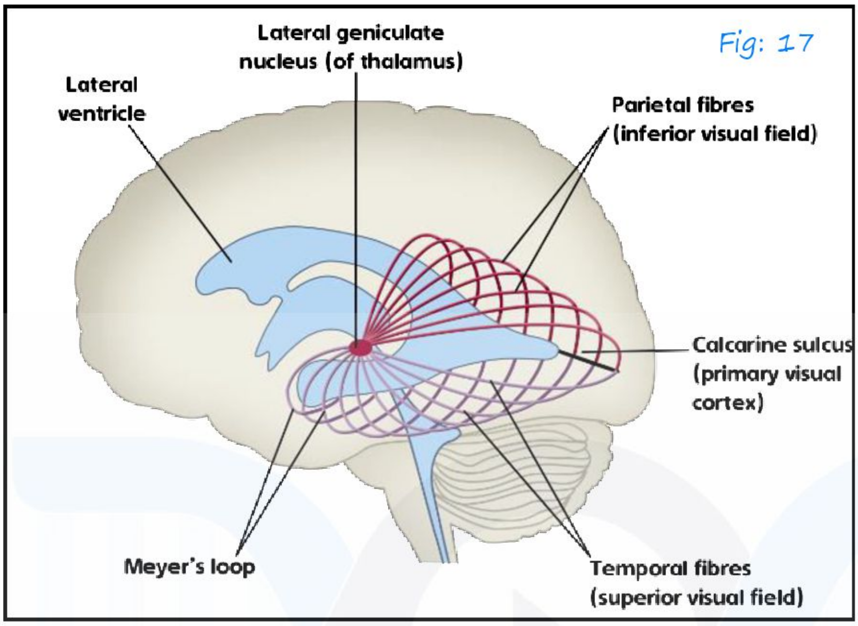

Lateral geniculate nucleus (of thalamus)
Lateral ventricle
Parietal fibres (inferior visual field)
Calcarine sulcus (primary visual cortex)
Meyer's loop
Temporal fibres (superior visual field)

Fig: 17

↓

i. Inferior fibers – Meyer– Temporal lobe

ii. Superior fibers – Baum's partial lobe

- Defect @ left Meyer loop → inferior fibers of
  - Left temporal ⎤
  - Right nasal ⎦ lost

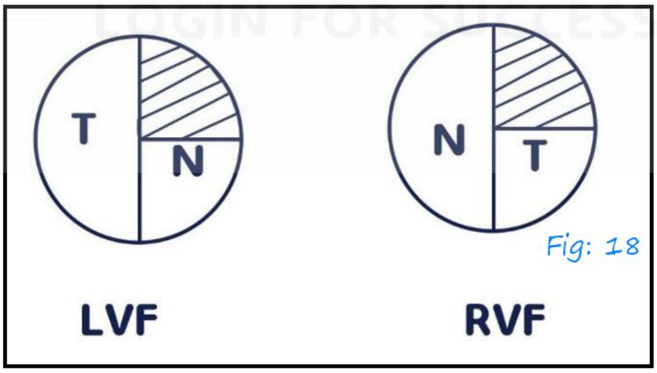

T
N
N
T
LVF
RVF

Fig: 18

↓ Quandrantopia homonymous superior hemianopia

(PIE on SKY)

- Defect Baum's loop PIE ON FLOOR

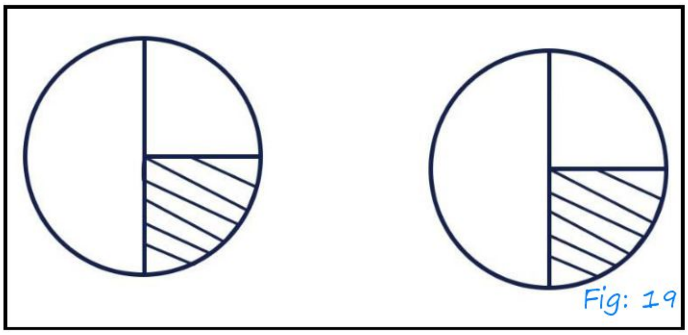


Fig: 19

Meyer- SIT

- Pie on sky
- Inferior fibers
- Temporal

- Pupil – normal
- No optic atrophies

Lesion @ visual cortex

- Visual cortex is primarily supplied by posterior cerebral artery→ Most common
- Tip is supplied by middle cerebral artery
- Right homonymous hemianopia→ macular sparing

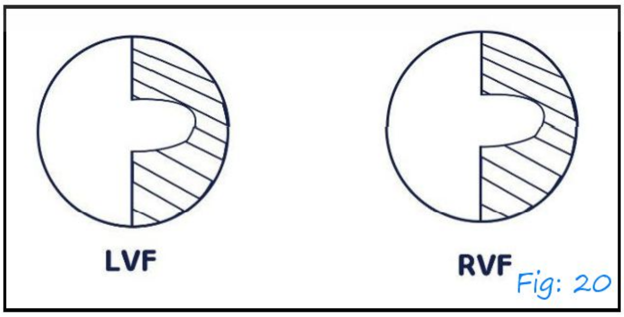
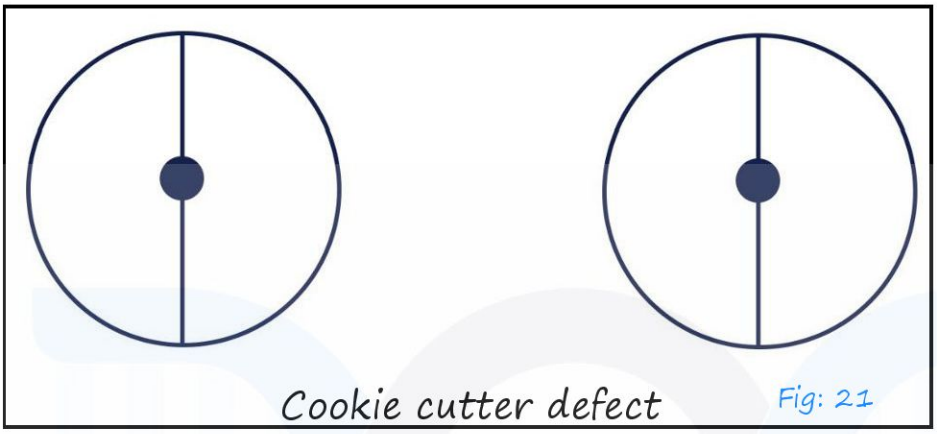
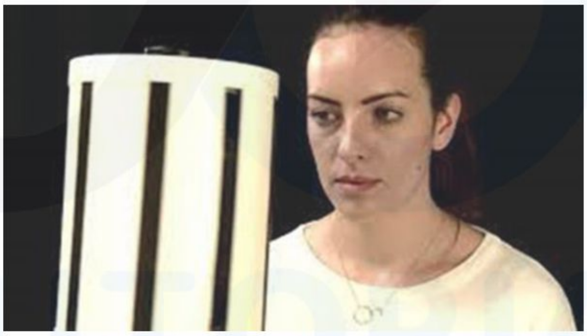


Fig: 20

- Since visual cortex receives much larger input from macular area, the macula fibers may less affected in lesions of occipital cortex
- If lesion involves only tip of cortex



Cookie cutter defect Fig: 21



OK drum Fig: 22

Homonymous hemianopia sand cocagan dictum

- Symmetrical OKN with homonymous hemianopia

  ↓

  Cortical, frontal lesion
- Asymmetrical OKN with homonymous hemianopia

  ↓

  Parietal lesion
- Symmetrical OKN is due to Parieto occipital visual area 18,19

Cortical Blindness 00:46:46

- B/L cortical infarct → B/L vision loss
  → Pupils Normal
- Anton syndrome: - denies blindness
- Riddoch phenomenon – filtered perception of moving objects

Lesions in pathway	Visual filed defect
• I/L optic nerve	• Anopia, central, centoceal
• Proximal part of nerve	• Junctional scotoma of tarquin
• Chiasma- centre	- Bitemporal hemianopia
- Peripheral	- Binasal hemianopia
- Craniopharyngioma	- Inferior quadrantanopia
- Pituitary Macroadenoma	- Super quadrantanopia
• Optic tract	• C/L homonymous hemianopia key hole object
• Lateral geniculate body	• Key hole defect
• Optic radiation	
- Temporal lobe	• Pie on sky
- Parietal	• Pie on sky
• Visual cortex	• Macular sparing
• Tip of cortex	• Cookie cutter defect

DOC TUTORIALS LOGIN FOR SUCCESS

Sphincter pupillae
↓
Constriction

Abnormal pupil 00:07:50

1. Amaurotic pupil
   - Also called as APD (Afferent pupillary defect)
   - Bilateral optic nerve lesion (chronic)
     ↓
     No constriction of light on either eye

2. RAPD (Relative afferent pupillary defect)
   (Marcus Gunn pupil)
   - Seen in U/L on disease
     Bilateral on disease → asymmetric

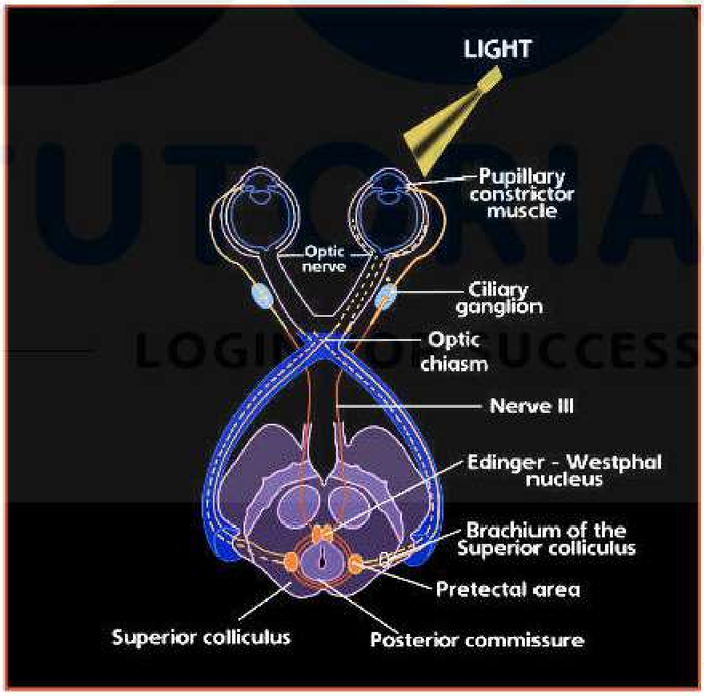

LIGHT
Pupillary constrictor muscle
Optic nerve
Ciliary ganglion
Optic chiasm
Nerve III
Edinger - Westphal nucleus
Brachium of the Superior colliculus
Pretectal area
Superior colliculus
Posterior commissure

Fig: 2

TUTORIALS
LOGIC OF SUCCESS

PUPILLARY PATHWAY AND ABNORMAL PUPILS

- Normal pupillary reaction
  - Direct light reflex
  - Consensual light reflex → indirect light reflex

  ] Pupil constriction to light
- Normally pupils also constrict on accommodation

Light reflex pathway (PIE – TICS) 00:01:55

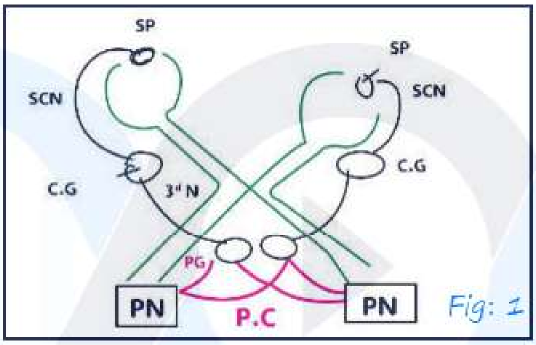


Fig: 1

Rods and cones
↓
Bipolar cells
↓
Ganglion cells
↓
Optic nerve → Afferent nerve
↓
Chiasma
↓
Pretectal nucleus
↓
Internuncial neurons
↓
Edinger Westphal nucleus
↓
3rd nerve → efferent nerve
↓
Inferior oblique
↓
Ciliary ganglion
↓
Short ciliary nerve
↓

Internuncial neurons:
- Most important structure for consensual light reflex
- Periaqueductal grey
- Posterior commissure

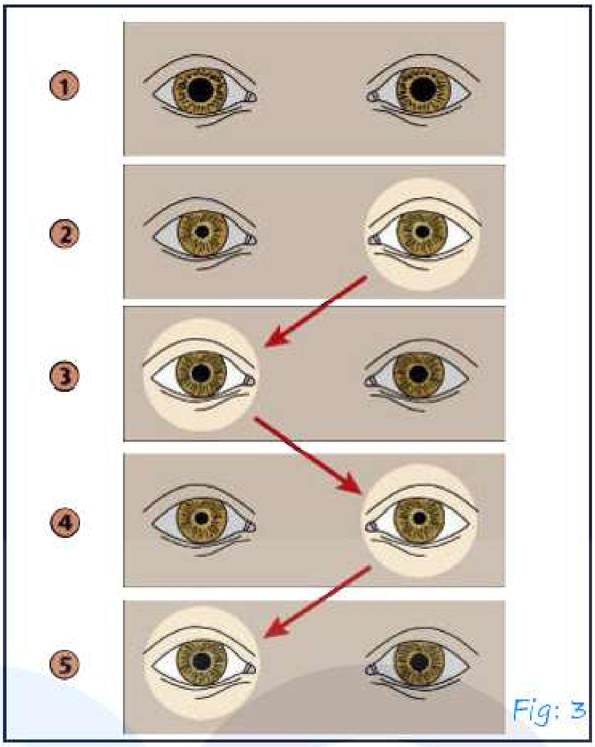


Fig: 3

Example: Right optic neuritis → loss of vision 1 day

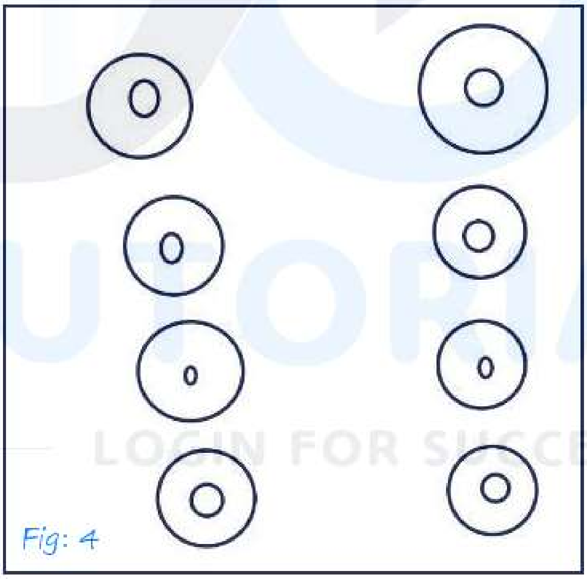


Fig: 4

- The moment I show torch of light on right side
  - Slight constriction of pupil
  - Direct and consensual light reflex gone
- Torch of light on other lift side

  ↓

  Good constriction of pupil

- Quickly shift the light to other side

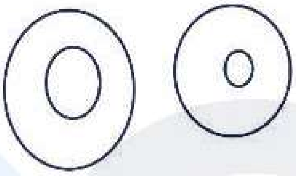
  ↓↓

  Relative dilatation of pupil

  ↓↓

  Paradoxical dilatation → Pupillary escape phenomenon

3. Tonic pupil



Light near dissociation

↓

- U/L dilated pupil → ANISCORIA
- light reflex absent
- Accommodation reflex ⊕ → tonic
- vermiform contraction

- occurs post viral infections when there is problem at ciliary ganglion
  - herpes
  - DM
- diagnose: Hypersensitive to dilute pilocarpine
  - denervation to cholinergic
  - diluted pilocarpine 0.125% → Brisk constriction of pupil
- association of absent deep tendon reflex

  ↓↓

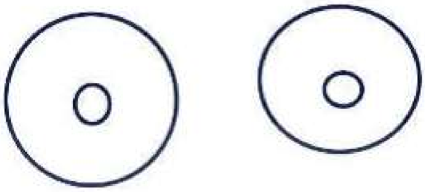
  Adie – tonic pupil

4. Argyll Robertson pupil

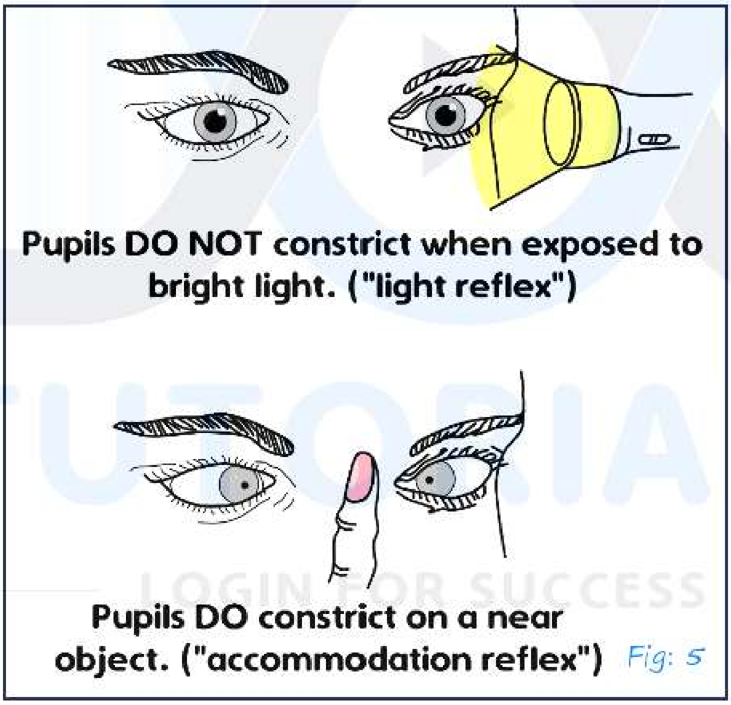
- Can be seen in syphilis / neurosyphilis
- Accommodation reflex present (ARP)
- Light reflex lost (LRL)

Light near dissociation

B/L small pupil



- Also called as prostitute pupil



Pupils DO NOT constrict when exposed to bright light. ("light reflex")

Pupils DO constrict on a near object. ("accommodation reflex") Fig: 5

DOC TUTORIALS
LOGIN FOR SUCCESS

5. Wernicke's hemianopia pupil

- Optic tract lesion
- Lesion in lift optic tract → torch of light in nasal area lift

↓

No light reflex

- Torch of light on temporal side

  ↓

  Constriction of pupil
- If affected hemiretina is stimulated → no pupillary response

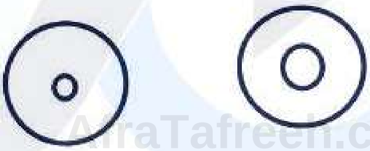
6. Hutchinson's pupil
   - 3rd nerve palsy
   - Head trauma

   a) Pupil small – irritation of 3rd nerve

   b) Pupil dilated U/L – paralysis of 3rd nerve

   c) Bilateral dilated pupil

7. Horner's pupil



AiraTafreen.com

- Paralysis of sympathetic nerve

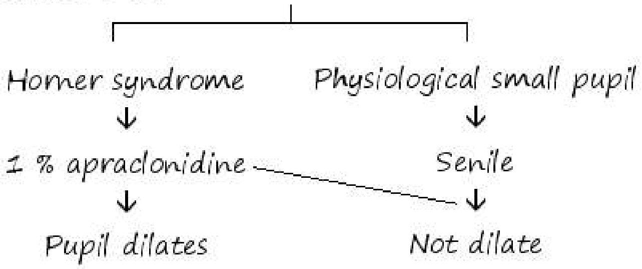
  ↓

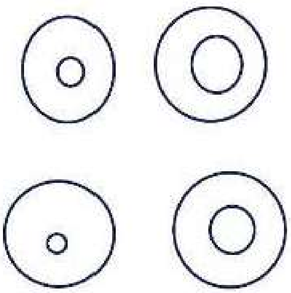
  No contraction of dilator pupillae
- Anisocoria increase in dark

TUTORIALS LOGIN FOR SUCCESS

Anisocoria 00:23:57

- Different size of pupil
- Significant if the difference > 0.4 mm
- If anisocoria increase in dark → small pupil is abnormal


Horner syndrome
Physiological small pupil
1 % apraclonidine
Senile
Pupil dilates
Not dilate



→ Bright light

→ anisocoria increase

↓

Dilated pupil abnormal

a. Tonic pupil – constricts on diluted pilocarpine

b. 3rd in palsy – constricts on 2% pilocarpine

c. Drug induced dilated pupil → not constricted by pilocarpine

Horner's syndrome 00:30:08

- Occulosympathetic paralysis
- HI – PAMELA
  - HI – Heterochromia iridium (Hypochromic iris)

    ↓

    Seen only in congenital cases
  - P – Ptosis → mild (Multer's muscle affected)

    ↑

    Sympathetic normal

    LPS normal
  - A – Anhidrosis → absence of sweating over ½ of face
  - M – MIOSIS → dilator pupillae muscle supplied but sympathetic normal
  - E – Enophthalmos → sunken eye (Ptosis of UL and LL)
  - L – loss of ciliospinal reflex
  - A – aniscoria – increase in dark.

SUPRANUCLEAR CONTROL OF MOVEMENTS

- Saccades – Quick refixation movement
- pursuit – Smooth defoviating following movement
- Fast → Frontal cortex – Fast – Saccade
- Slow → Parieto occipital area → Pursuit

Horizontal gaze centre

- Frontal eye field (FEF) (Most important)
- PPRF → Pontine paramedian reticular formation

Pathway of horizontal gaze

- RFEF → Gets innervation to PPRF on (L) side

↓

6th nerve

↓

6th supplies LR

- PPRF → Also gives innervation to 3rd nucleus

↓

3rd nerves innervate MR

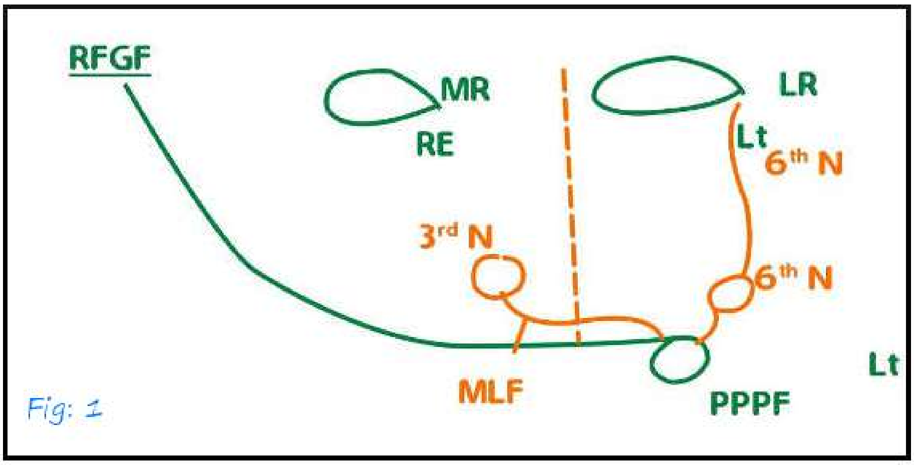


Fig: 1

Horizontal gaze palsies 00:07:56

- Lesion on PPRF
- Problem on (L) PPRF → I/L gaze palsy
- Lesion on FEF
- Problem in left MLF → left adduction gone & Right adduction (+) → Ataxic nystagmus →(INO) Inter nuclear ophthalmoplegia
- Combined MRF and PPRF defect
- Problem in Right
- Right gaze defect
- Right adduction faulty
- Abduction of ZE only preserved with ataxic type of nystagmus
- ½ syndrome
- 8½ Syndrome
- 7th nerve palsy combines with in ½ syndrome

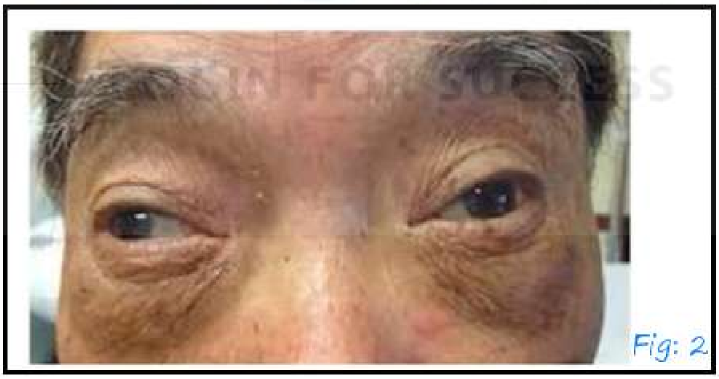


Fig: 2

- Bilateral MLF affected → WEBINO wall eye bilateral intra nuclear ophthalmoplegia → Convergence normal

Vertical gaze centre 00:15:41

- Rostral interstitial nucleus of MLF
- Posterior commissure

Disorder of vertical gaze

- Perinaud – Dorsal midbrain syndrome
- Lesion at posterior commissure
- C/F vertical gaze palsy
- Gradually ophthalmoplegia
- Light near dissociation
- Collier sign – Lid retraction
- Convergence retraction nystagmus

Progressive supranuclear palsy

- Elderly males
- Downgaze palsy → 1st → Upward gaze → Global akinesia
- Ataxia (+)
- Extrapyramidal sign (+)

DX TUTORIALS LOGIN FOR SUCCESS

kearn Sayre syndrome

- Most severe type
- Chronic progressive external ophthalmoplegia (PED)
- Single sporadic deletion on M-DNA
- Associated with cardiac conduction defects & Pigmentary retinopathy

- Variable ptosis
- Diplopia
- ICE pack test → Improvement of ptosis

  ICE pack is placed on eye for 5mins
  ↓
  Low temperature decreases acetylcholinesterase
  ↓
  Improves ptosis
- FAN test → Looks up towards fan
  ↓
  Gradual drooping

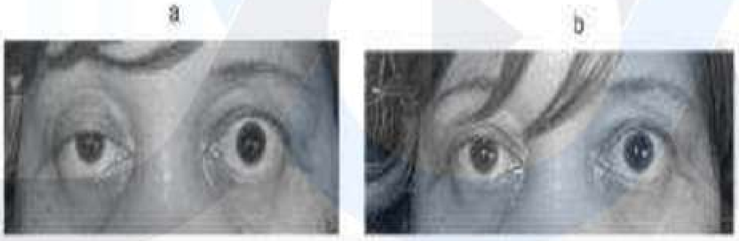


Fig: 3

Edrophonium test
↓
After edrophonium
↓
Ptosis improves

Nystagmus 00:23:51

- Regular repetitive rhythmic to and fro movement of eye

Types

- Pendular
  - ↓ Equal amplitude and velocity on both sides
- Jerk
  - Fast phase
    - Right beat
    - Up beat
    - Left beat down
  - Slow phase

- Optokinetic nystagmus → pathway → parieto – Occipital area
- Rail road nystagmus
- Elicited by
- Jerk nystagmus
- Pathway
- If drum left to right and saccade right to left
- Vestibular ocular reflex COWS → Cold water – Opposite & Warm water → same
- End gaze → By cooking at extreme of gaze
- Null zone → Nystagmus minimal and vision better acquired nystagmus

Down beat

- C – Cervico medullary junction
- A – Arnold Chiari malformation
- D – Down beat

Up beat

- P – Pontine lesions
- P – Posterior fossa tumor
- V – Vermis of cerebellum
- Phenytoin induced nystagmus in up gaze only

Convergence retraction → Lesion in posterior commissure

- Pineal gland tumor
- Perinaud's syndrome
- Looks up – Bilateral MR contracts → Retraction of globe

See-Saw

- One eye goes up and other down ↑intort ↓extort
- Seen in pituitary gland tumor – Parasellar region
- Miner's nystagmus – Dark induced
- Ataxic nystagmus – Seen in IND lesion in MLF
- Spasmus nutans classically characterised by the triad of binocular small amplitude pendular nystagmus → Head nodding & Torticollis

COLOR VISION

- Genes for Red- green located on X chromosome
- Gene for Blue colour located on 7 chromosomes → AD Tritanopia
- Primary colours → Red, green and blue
- Most sensitive colour → Green
- Red → Seen from far

Cones → 3types

- S cones – Short wavelength cone & Perceive Blue colour
- M cones – Middle wavelength cones & Perceive Green colour
- L cones – Long wavelength cones & Perceive Red colour

TUTORIALS LOGIN FOR SUCCESS

Colour blindness 00:02:53

- 2 types
- Congenital
- Acquired

Congenital

- Most common
- Red green more common than blue
- Green is the individual most common colour defect

Acquired

- Optic nerve problems, macular problems → Blue colour defect generally
- Oldage → Blue blind
- Normally we are Trichomat → 3 colour perception
- Monochromat – One single colour perception
- Dichromat – 2 color perception
- Can be
  - Protonopia → Red absent
  - Deutranopia → Green absent
  - Tritanopia → Blue absent
- Trichomats can be
  - Protanomaly → Red color defect
  - Deutranomaly → Green color defect
  - Tritanomaly → Blue color defect

TUTORIALS LOGIN FOR SUCCESS

Tests for color vision 00:07:23

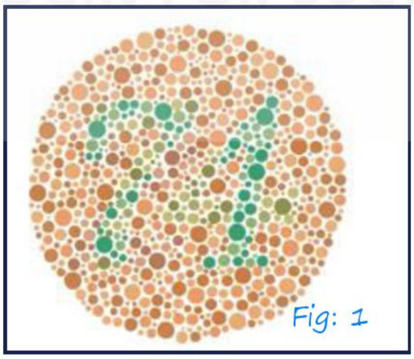


Fig: 1

1.Ishihara chart

- 38 total test plates
- Has numerals written

- Have to check by closing eye
- First chart can be seen by color-blind patient also
- Good screening test
- Problem → Only tests red and green color

2.Lantern test

- Done for railway employs

3.City university test

Fig: 2

- 10 test plates in form of dots

4.Hardy rand ritter test

- 24plates
- Shapes (+)

5.Anamoloscope

- Sensitive test
- Difficult to be done and to interpretation

Fig: 2

6.Farnsworth Munsell 100 HUE test

- 85 Hue tested
- Most sensitive

7.FM 15th Hue test

BASICS EXTRAOCULAR MUSCLES AND ACTIONS

- Extraocular Muscles: Muscles outside eye (Total 7 in numbers)
  - Recti-4
  - Oblique-2
  - LPS
- Intraocular Muscles: - ciliary Muscle
  - Iris Muscle (sphincter pupillae & Dilator Pupillae)

Origin and Insertions of EOM

	Origin	Insertion
Superior Rectus	Annulus of Zinn	On sclera close to limbus anterior to equator (imaginary plane 14 mm behind limbus)
Inferior Rectus	Annulus of Zinn	On sclera close to limbus anterior to equator (imaginary plane 14 mm behind limbus)
Medial Rectus	Annulus of Zinn	On sclera close to limbus anterior to equator (imaginary plane 14 mm behind limbus)
Lateral Rectus	Annulus of Zinn	On sclera close to limbus anterior to equator (imaginary plane 14 mm behind limbus)

Superior oblique Longest & thinnest EOM	Body of spheroid From trochlea it takes oblique course & meet on sclera trochlea is believed to be true origin. (A/K/A Muscle pulley)	On sclera posterior to equator
Inferior oblique	Only muscle originated from Anterior part of orbit (i.e., orbital surface of maxilla) it passes under inferior Rectus	On sclera Behind equator Near insertion of vortex vein
LPS	Lesser wing of sphenoid	Upper layer of tarsal plate

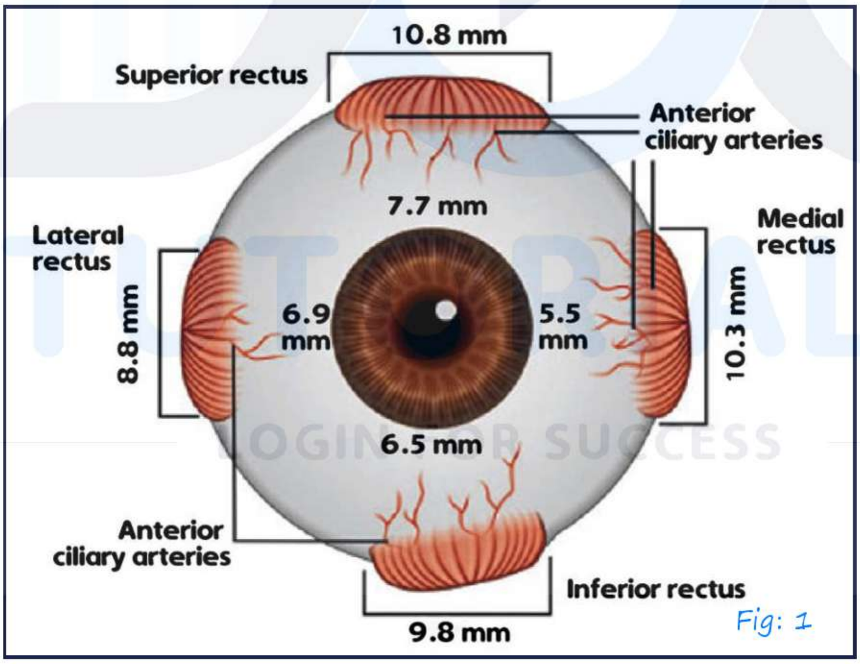

10.8 mm
Superior rectus
Anterior ciliary arteries
7.7 mm
Lateral rectus
Medial rectus
8.8 mm
6.9 mm
5.5 mm
10.3 mm
6.5 mm
Anterior ciliary arteries
Inferior rectus
9.8 mm

Fig: 1

Actions of EOM 00:14:15

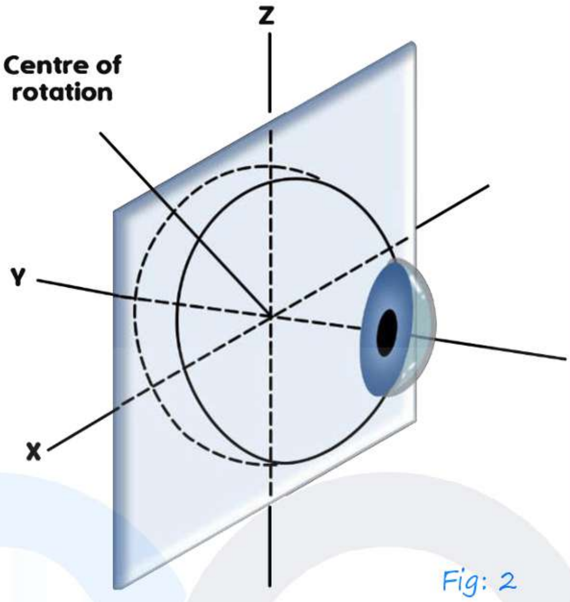
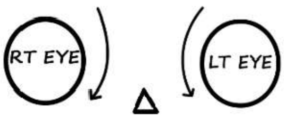
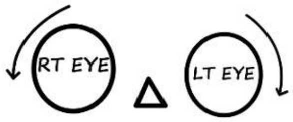

Z
Centre of rotation
Y
X

Fig: 2

Movements

i.DUCTION	Uniocular Movements	
Supraduction	Upward Movements	
Infraduction	Downward movement	
Abduction	Outward movement	
Abduction	Inward movement	
Intorsion	Rotation towards nose It is along Y axis	 RT EYE LT EYE
Extorsion	Outward rotation away from Nose It is along Y axis	 RT EYE LT EYE

ii. VERSION	Binocular simultaneous movement in same visual direction	+ Primary gaze→ 9 directions of gazes
Supervision	Dextro elevation	
Infra version	Dextro depression	
Dextro depression	Levo depression	
Levo version	Levo elevation	

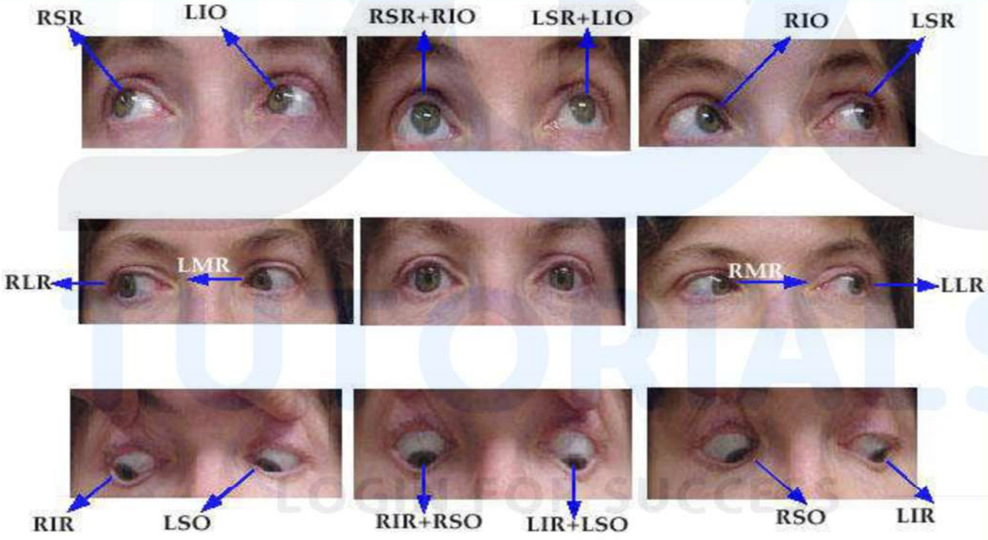

RSR
LIO
RSR+RIO
LSR+LIO
RIO
LSR
RLR
LMR
RMR
LLR
RIR
LSO
RIR+RSO
LIR+LSO
RSO
LIR

Fig: 3

Vergence

- Binocular movement in opposite Direction
- Convergence: Both eyes moving in
- Divergence: Both eyes moving out

Action	Primary	Secondary	Tertiary
MR	Adduction	–	–
LR	Abduction	–	–
SR	Elevation	Intorsion	Adduction
IR	Depression	Extorsion	Adduction
SO	Intorsion	Depression	Abduction
IO	Extorsion	Elevation	Abduction

Mnemonics

- S-IN→ superior – intorters (∴ inferiors are extorters)
- R- AD→ Recti- adductors (except lateral Rectus which is abductor)
- SO- ABID→ superior oblique (abduction, introrter, depressor)

Note

- Superior Rectus & Inferior Rectus makes angle of 23° with visual axis

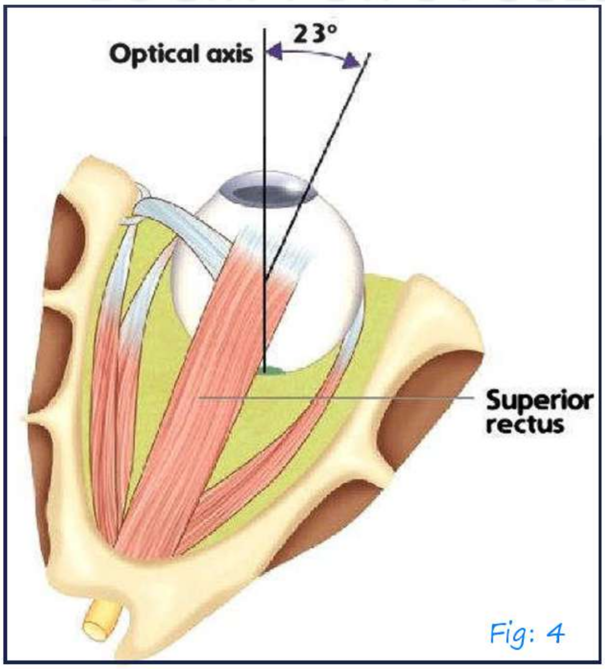


Fig: 4

- Superior & inferior oblique make an angle of 51° with visual axis

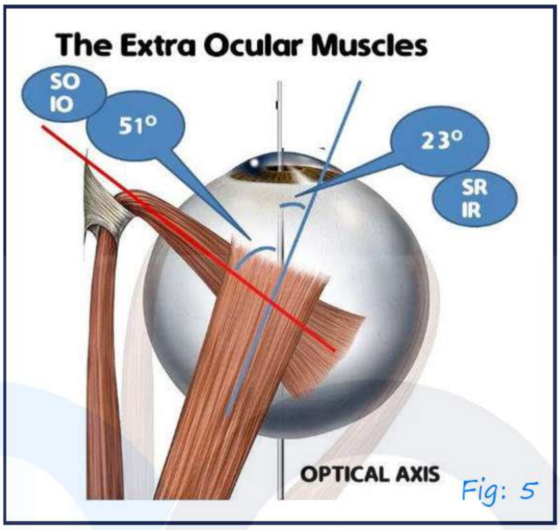


Fig: 5

Primary Action

- Primary action of vertical Muscles is in Abducted eye

  ∴ In Abducted position visual axis & SR are in parallel direction

  ∴ In this position it acts as pure elevator
- Similarly inferior Rectus in this position will act as pure depressor
- Superior oblique become i. chief intorter in abduction

  ∴ Muscle & visual axis will be perpendicular

  ii. pure Depressor in adduction

  ∴ Muscle & visual axis will be in parallel position

Note

- Primary actions of for Superior & inferior Muscle are in abduction
- Secondary actions of Superior & inferior Muscles are in adduction

Clinical Action of Muscle 00:33:42

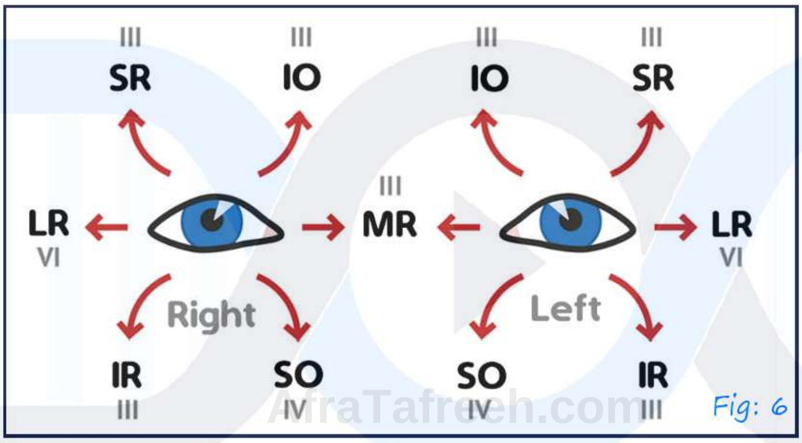
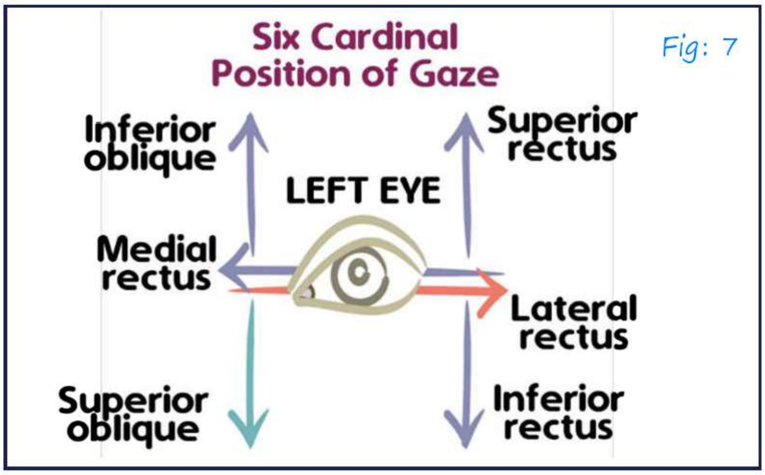

III
SR
III
IO
III
IO
III
SR
LR
VI
III
MR
LR
VI
Right
Left
IR
III
SO
IV
SO
IV
IR
III

Fig: 6

- Superior oblique- pure Depressor in adducting Eye
- Inferior Rectus- pure Depressor in abducting Eye

Cardinal Position of Gaze 00:37:30

- Six cardinal positions of Eye gaze isolate the individual EOM
- It helps to determine which Muscle in Abnormal


Six Cardinal Position of Gaze
Fig: 7
Inferior oblique
Superior rectus
LEFT EYE
Medial rectus
Lateral rectus
Superior oblique
Inferior rectus

Herring's Law

- Equal innervation goes to a set of yoke Muscle
- Eg Rt LR & Lt MR has equal innervation

Sherrington's law

- Equal but opposite innervation goes to agonist & antagonist Muscle

Terminologies for Muscle

- Agonist: the muscle which is responsible for movement

  e.g., for Rt abduction – Rt LR
- Antagonist: The Muscle with opposite ation to the movement occurring

  e.g. Rt MR in Rt abduction
- Yoke Muscle: Muscle in each that accomplish given version

  e.g., Rt Lr & Lt MR in dextroversion
- Synergists: The Muscle causing same action

  e.g., supraduction – SR
  IO

TUTORIALS LOGIN FOR SUCCESS

DEFINITION AND TYPES OF SQUINT AND BINOCULAR VISION

- Also known as: strabismus
- Deviation of Either eyes I called squint

Types

- Pseudo squint: Apparent squint, visual axis are parallel

  e.g., Epicanthic fold – extro lid skin near Medial canthus

  Types T- Epicanthus Tarsalis
  I- Epicanthus Invereus
  P- Epicanthus Palpebralis
  S- Epicanthus Superciliaris

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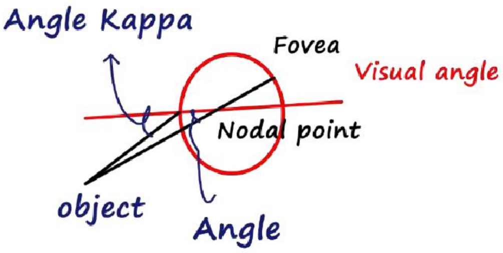
Other causes

- Mnemonic: NAK E MT- pseudoesotropia
  - i. Negative angle kappa
  - ii. Epicanthic fold
  - iii. Myopic
  - iv. Telecanthus


Angle Kappa
Fovea
Visual angle
Nodal point
object
Angle

- Kappa – Nasal corneal Reflection
  - Normally it is Positive
- If fovea goes nasally than kappa becomes – negative normally8 it is located temporally

Pseudo Exotropia

- Pseudo Divergen squint
- Seen in – Positive angel kappa
  - Hyperdorism
  - Hypermetropia

PHORIA:

- Hidden squint

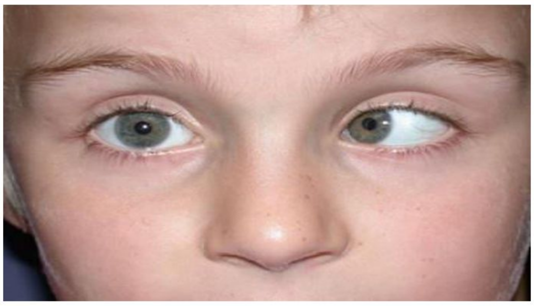
  i.e., here tendency to squint is present by is hidden by power fusion

↓

Become manifest squint during old age or excessive tireds

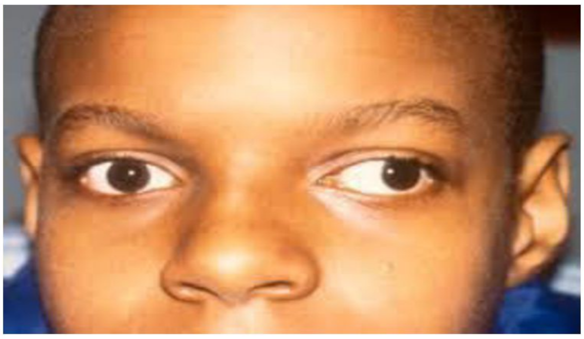
TROPIA

- Manifest squint
  - Esotropia – Inward Deviation of eye



"Convergent squint" Fig: 1

- Exotropia – Outward Deviation of Eye



"Divergent squint" Fig: 2

- Hypertropia – Upward Deviation of Eye

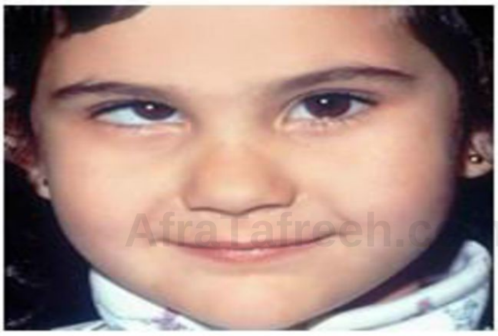
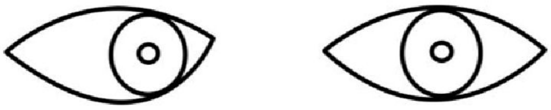


Fig: 3

- Hypotropia – Downward Deviation of Eye

Phenomenon 00:11:30

- Why squint Happen?



i. Rt LR is not contracting

ii. 6th Nerve /Nuclear damages

iii. Rt MR is fibrotic – not able to Relax

Binocular Vision 00:13:30

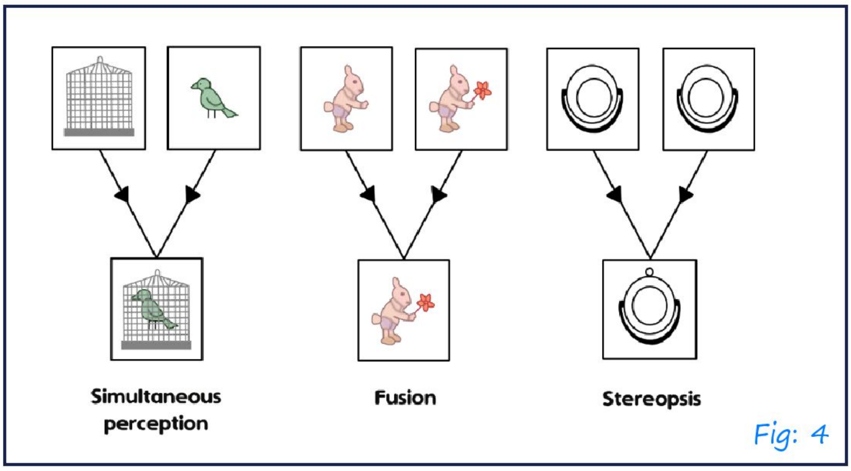

Simultaneous perception
Fusion
Stereopsis

Fig: 4

i. Simultaneous (Grage1) Perception n- Ability of Both eyes to perceive simulation tow images, one formed on each Retino

ii. Fusion – Process ability to fuse two images in one single frame

iii. Steroids – Most advanced grade

– 3rd Dimension → Ability to perceive Depth

Normal Retinal Corresponding Points 00:16:35

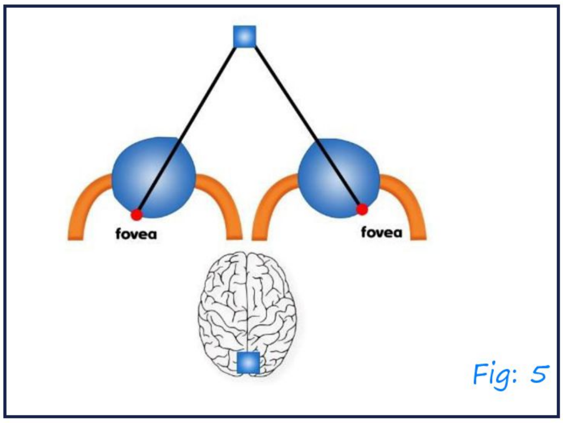

fovea
fovea

Fig: 5

- Point on Retina that give rise to same Visual Direction are called Retinal corresponding points

PROBLEMS IN SQUINT

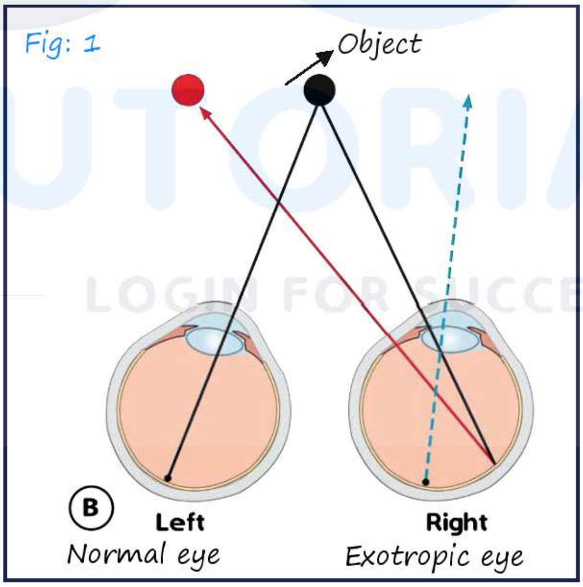
Problems with squint

- There are two types of problems → I. Diplopia
  II. Confusion

I. Diplopia

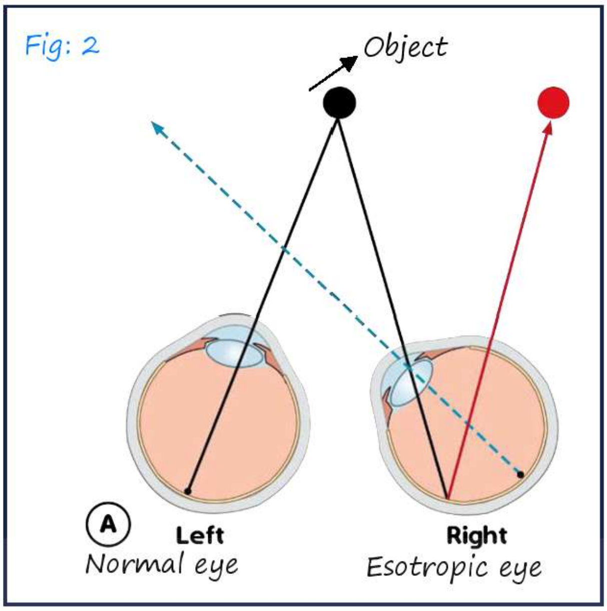
- Diplopia is the simultaneous perception of two images of a single object
- Image of same object falling on non-corresponding points on retina
- Diplopia is of two types – Crossed
  – Uncrossed
- Crossed Diplopia – Seen in Exotropia

Fig: 1


Object
B
Left
Normal eye
Right
Exotropic eye

- Uncrossed diplopia – seen in Exotropia

Fig: 2


Object
A
Left
Normal eye
Right
Esotropic eye

II. Confusion

- Image of two different objects are formed at 2 fovea

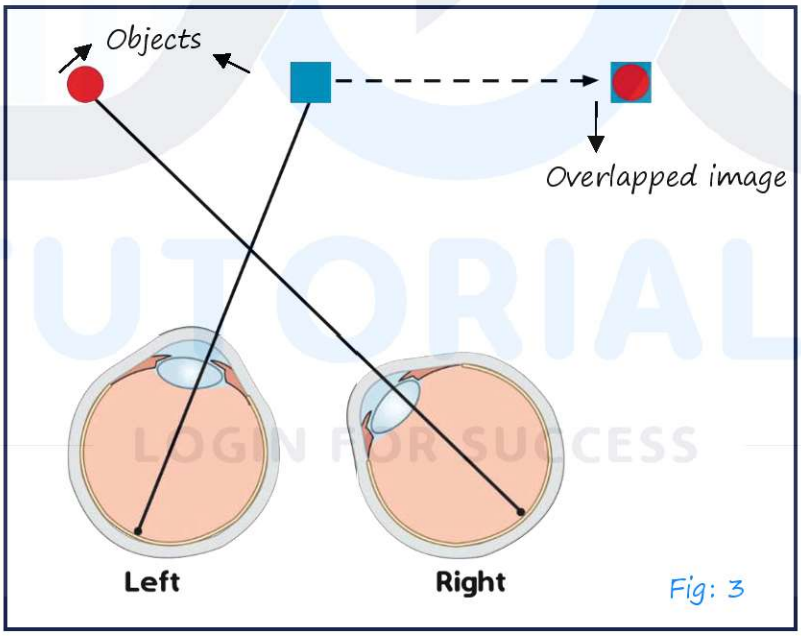

Objects
Overlapped image
Left
Right

Fig: 3

TUTORIALS LOGIN FOR SUCCESS

Adaptations to Strabismus 00:08:10

- Three types

i. Abnormal Retinal Correspondence

- If there is a small angle squint, fovea of one eye behaves as corresponding point to a non-foveal point of another eye.

ii. Suppression

- It is helpful- when there is large angle squint
- Cortical suppression – suppress image formed by deviated eye (poor image)

iii. Abnormal Head Posture

- Motor adaption
  - Head turn /tilt
  - Face turn
  - Chin elevation/Depression

TESTS FOR SQUINT

Bruckner test 00:00:25

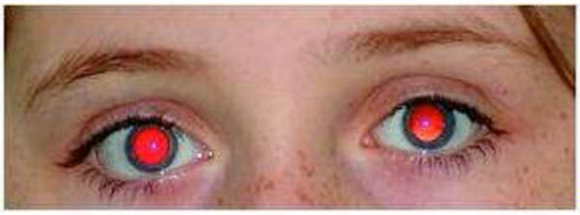


Fig: 1

- Normal red glow

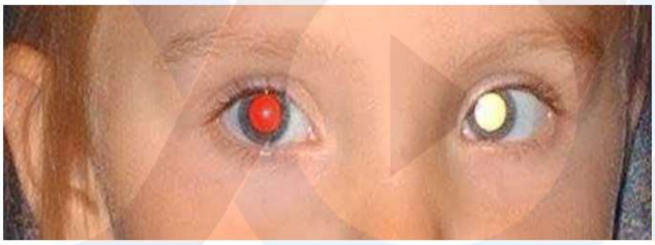


Fig: 2

- Lighter, brighter – poor vision of left eye
- RPE atrophy /any other problem of retina

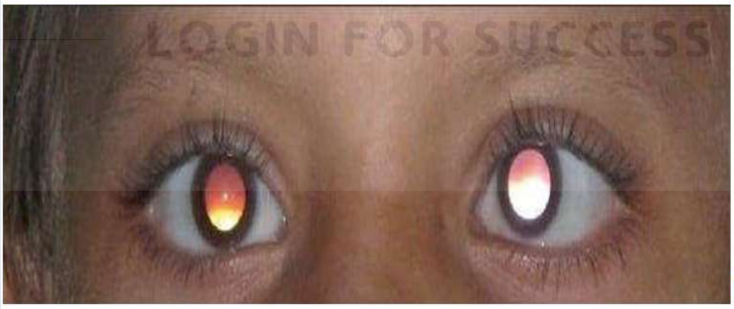


Fig: 3

- Crescents are seen – suggestive of refractive error

Hirschberg test 00:02:35

- Important clinical test
- Gives an estimation of amount of squint
- When a light is made to fall on eye, the reflection is seen in the pupillary area, which is in central – theoretically but actually it is slightly nasal it is because of positive angle kappa

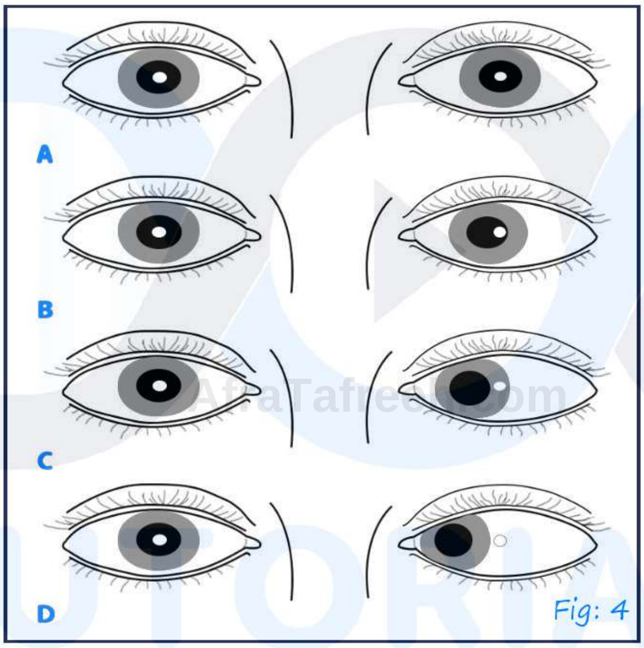


Fig: 4

- If there is a 1mm deviation of reflex the deviation of eye is by 7° = 14prism diopters (PD)
- 1° = ~2PD

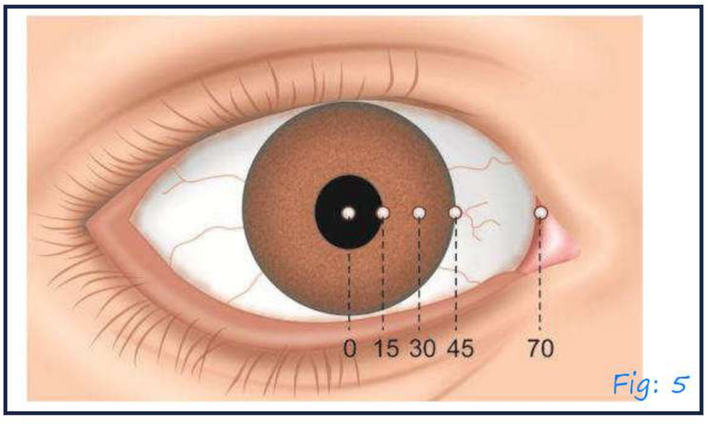
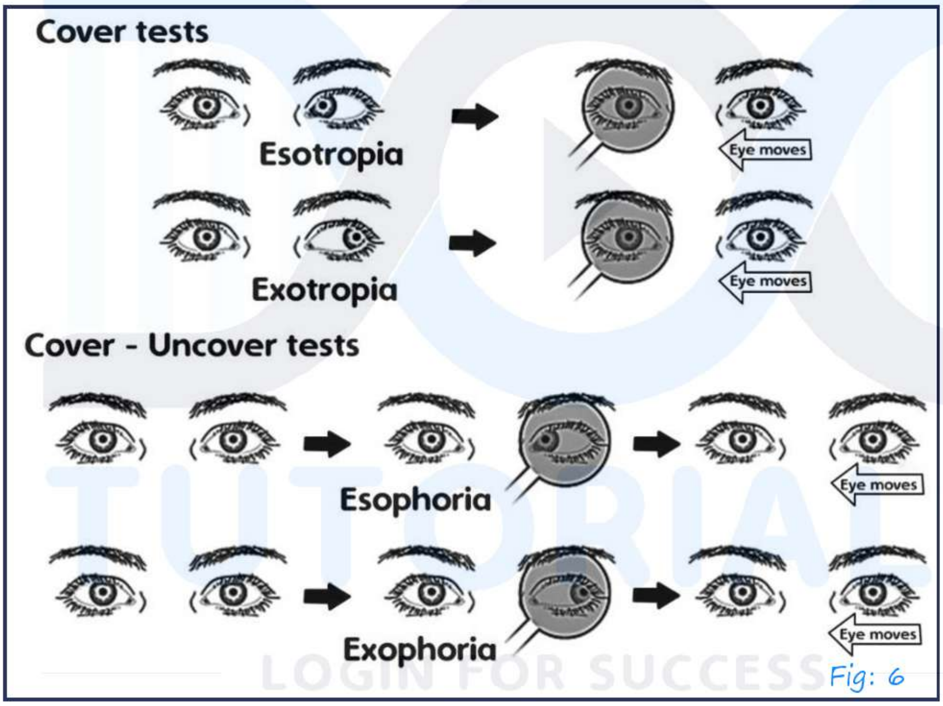


Fig: 5

- If deviation is 2mm from central reflection i.e, at pupillary border
- Squint = 15° = ~30PD
- If reflection is at limbus → 45° = ~90PD

Cover tests 00:08:20

- Cover – Uncover test


Cover tests
Esotropia
Eye moves
Exotropia
Eye moves
Cover - Uncover tests
Esophoria
Eye moves
Exophoria
Eye moves

TUTORIALS LOGIN FOR SUCCESS

Fig: 6

a. Cover test – Confirms tropia
   - Cover apparently normal eye
   - Refixation movement to centre of deviated eye

b. Cover – Uncover test – Confirms phoria

c. Alternate cover test
   - Total amount of squint can be known

Prism bar cover test 00:14:08

- Prism apex is placed at side of deviation

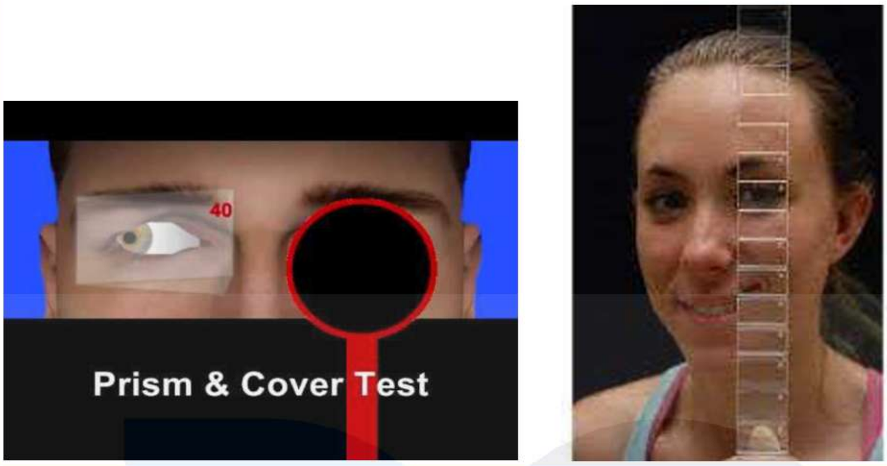


Fig: 7

- PBCT gives total actual amount of squint in prism diopters

Krimsky 00:17:25

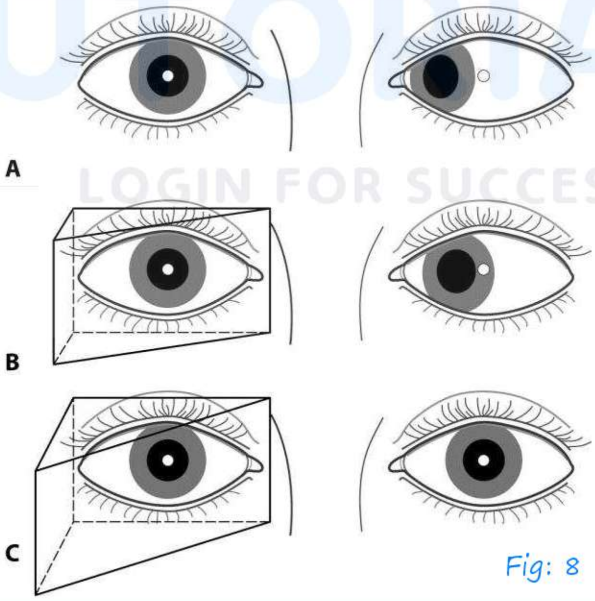


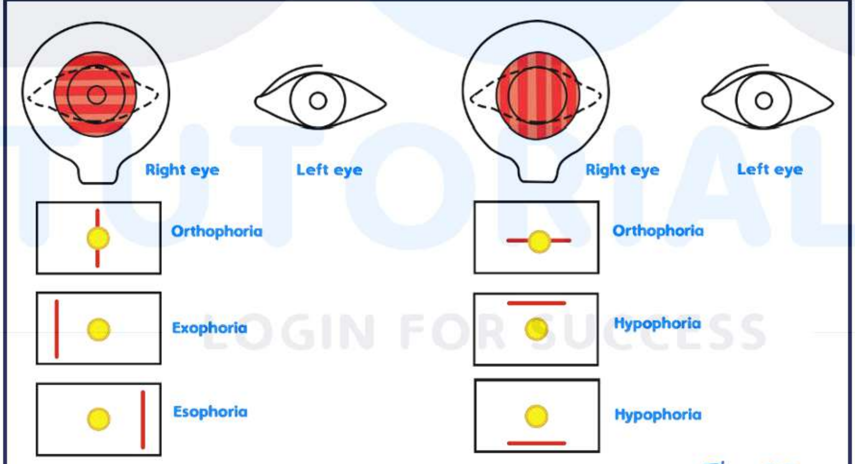
Fig: 8

- Here prism is placed over non deviating eye/apparently normal eye

Maddox rod test 00:18:18

Fig: 9

- It converts the point source of light into a line which is perpendicular to orientation of cylinder
- Breaking the fusion


Right eye
Left eye
Right eye
Left eye
Orthophoria
Orthophoria
Exophoria
Hypophoria
Esophoria
Hypophoria

Maddox rod test for horizontal and vertical heterophorias

Fig: 10

- Orthophoria – Straight eye
- It is generally done at a distance

Maddox wing test 00:23:47

- It is done at near like 33cm
- To check phorias

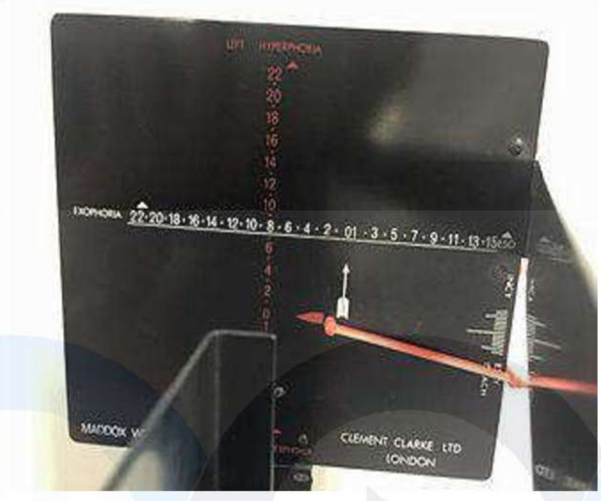

LEFT HYPERPHORIA
22
20
18
16
14
12
10
EXOPHORIA
22·20·18·16·14·12·10·8·6·4·2·01·3·5·7·9·11·13·15
MADDOX W
CLEMENT CLARKE LTD
LONDON

Fig: 11

- Right eye only sees white & red arrows
- Left eye sees only numbered line
- In normal individuals arrows point to '0'

Worth four dot test 00:25:26

- It is done to check binocularity

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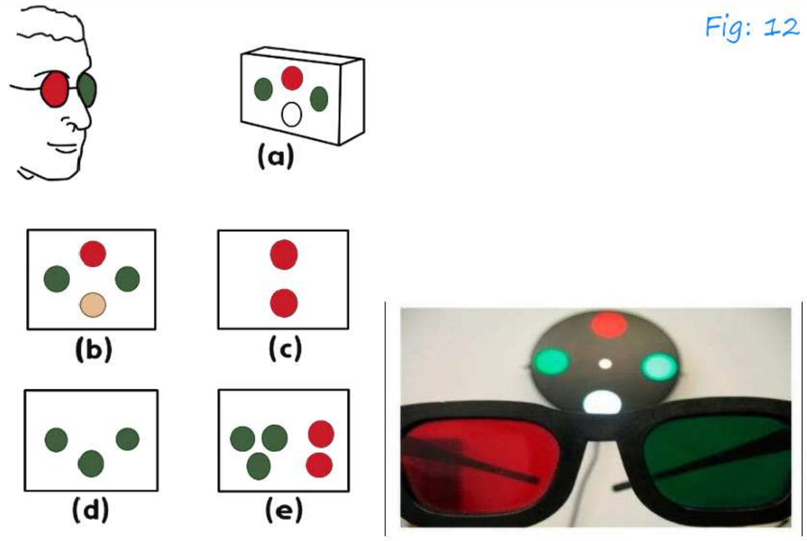
TUTORIALS
LOGIN FOR SUCCESS

Fig: 12



- Orthophoric – 1red, 2 green, 1 composite colour
- 2red – left suppression
- 3green – right suppression
- 3 green, 2red – Dipolopia

Stereopsis 00:28:58

- 2 pen test

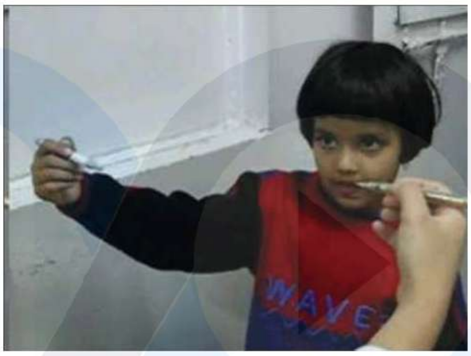


Fig: 13

- Tritmus fly test

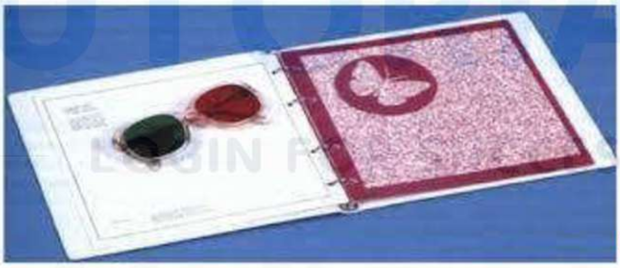


Fig: 14

- It is based on polaroid vectograph

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Synaptophore

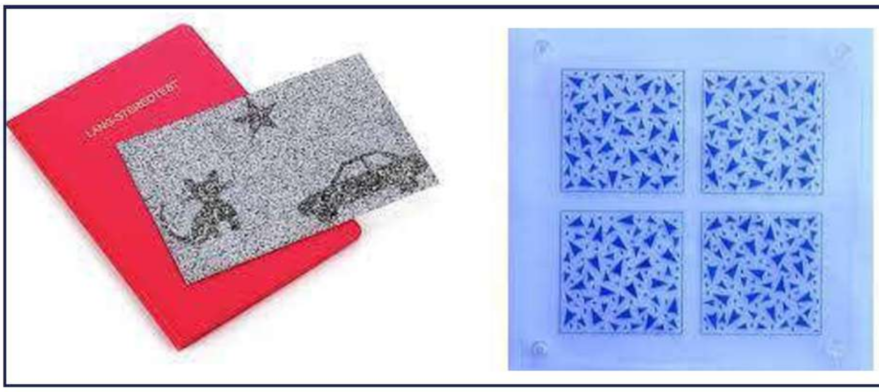
Fig: 15

Able to measure

- Grades of binocularity
- Fusion reserve
- Heterophoria
- actual amount of squint
- Orthoptic exercises – increase fusional capacity

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Forced duction test 00:34:13

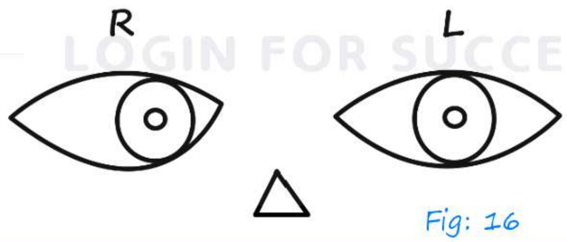

Fig: 16

- LR muscle paralysed – 6th nerve – paralytic squint or MR restricted
- Procedure – After applying anesthetic drops → Hold the limbus with lims forceps → try to move the eye

- negative forced duction test → Eye is moved easily → paralysis
- Positive forced duction test → Eye is not moved easily restrictive squint

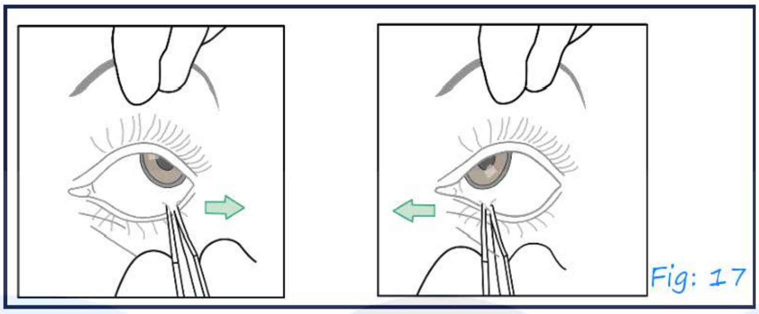

Fig: 17

Active force generation test 00:36:55

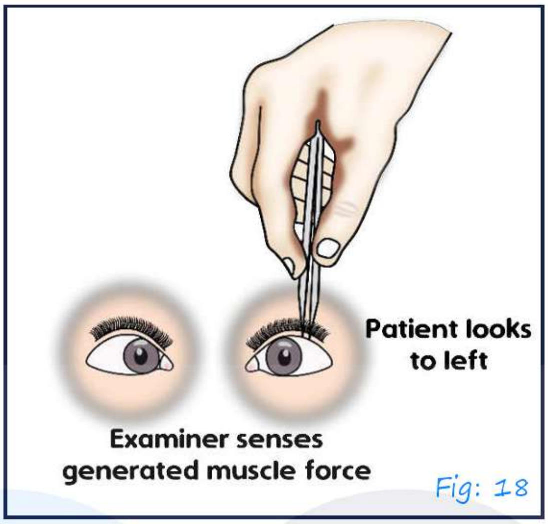
Patient looks to left
Examiner senses generated muscle force

Fig: 18

- Limbus is held with forceps
- Patient tries to move the eye

If eye does not move → Nothing felt by examiner → Paralytic

Eye is able to move → TUG is felt by examiner → Restrictive

TESTS FOR SQUINT

Bruckner test 00:00:25

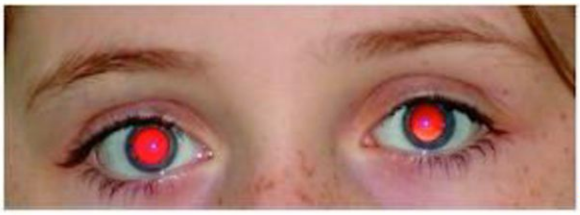
Fig: 1

- Normal red glow

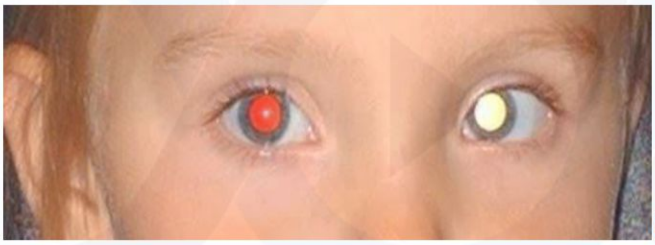
Fig: 2

- Lighter, brighter – poor vision of left eye
- RPE atrophy /any other problem of retina

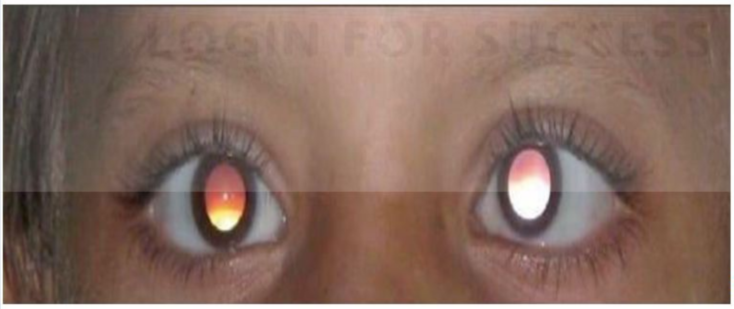
Fig: 3

- Crescents are seen – suggestive of refractive error

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Hirschberg test 00:02:35

- Important clinical test
- Gives an estimation of amount of squint
- When a light is made to fall on eye, the reflection is seen in the pupillary area, which is in central – theoretically but actually it is slightly nasal it is because of positive angle kappa

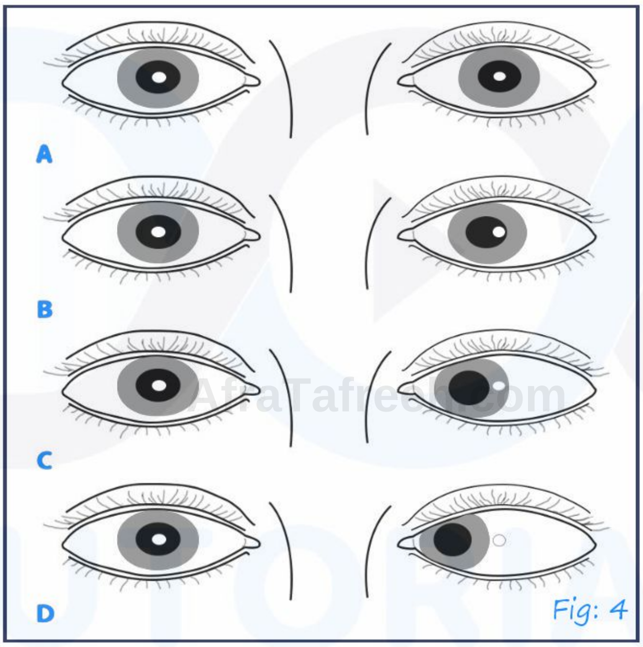

Fig: 4

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- If there is a 1mm deviation of reflex the deviation of eye is by 7^{o} = 14prism diopters (PD)
- 1^{o} = ~2PD

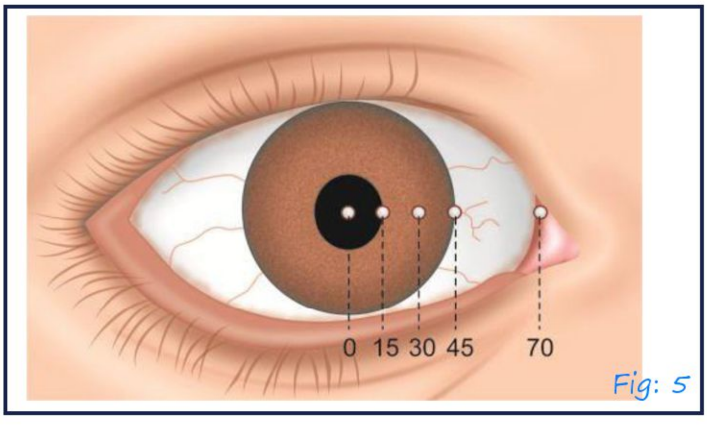


Fig: 5

- If deviation is 2mm from central reflection i.e, at pupillary border
- Squint = 15° = ~30PD
- If reflection is at limbus → 45° = ~90PD

Cover tests 00:08:20

- Cover – Uncover test

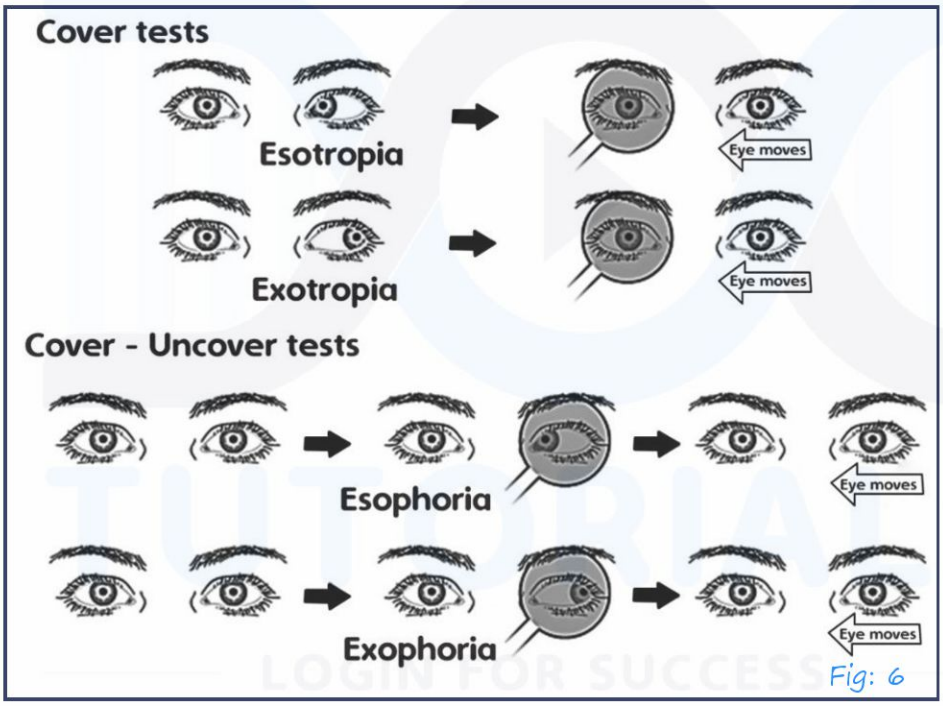

Cover tests
Esotropia
Eye moves
Exotropia
Eye moves
Cover - Uncover tests
Esophoria
Eye moves
Exophoria
Eye moves

Fig: 6

a. Cover test – Confirms tropia
   - Cover apparently normal eye
   - Refixation movement to centre of deviated eye

b. Cover – Uncover test – Confirms phoria

c. Alternate cover test
   - Total amount of squint can be known

Prism bar cover test 00:14:08

- Prism apex is placed at side of deviation

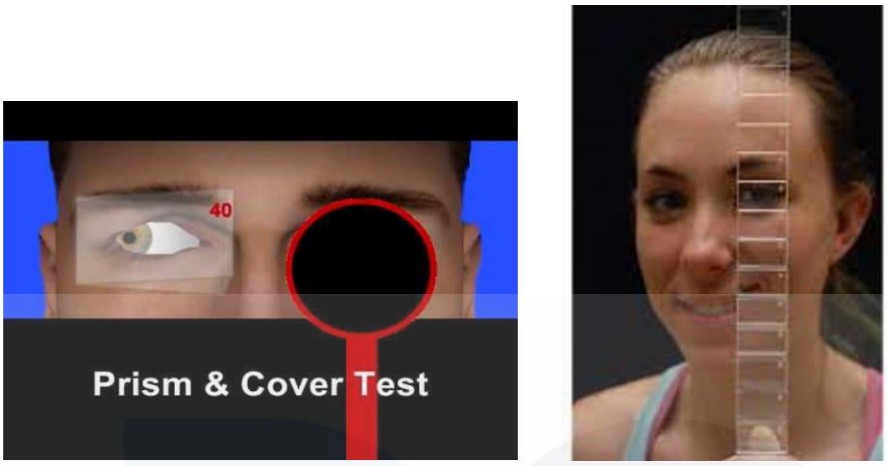


Fig: 7

- PBCT gives total actual amount of squint in prism diopters

Krimsky 00:17:25

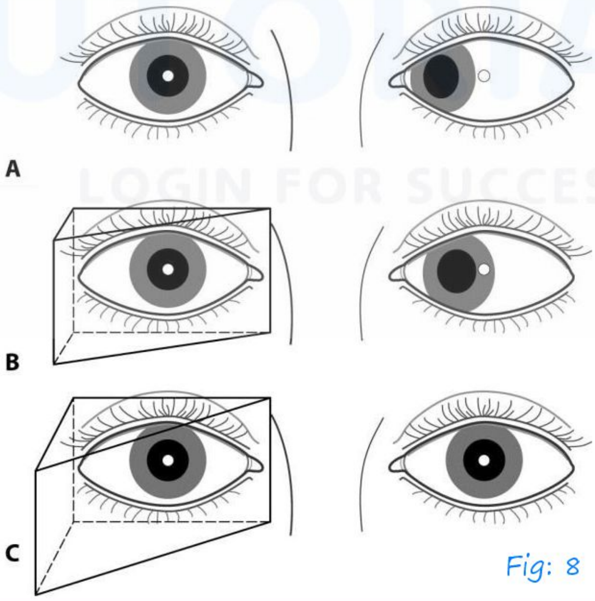


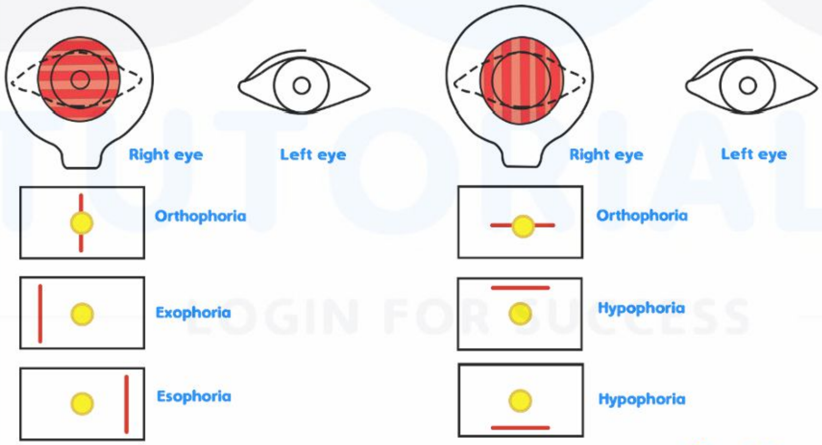
Fig: 8

- Here prism is placed over non deviating eye/apparently normal eye

Maddox rod test 00:18:18

Fig: 9

- It converts the point source of light into a line which is perpendicular to orientation of cylinder
- Breaking the fusion


Right eye
Left eye
Right eye
Left eye
Orthophoria
Exophoria
Esophoria
Orthophoria
Hypophoria
Hypophoria

Maddox rod test for horizontal and vertical heterophorias

Fig: 10

- Orthophoria – Straight eye
- It is generally done at a distance

Maddox wing test 00:23:47

- It is done at near like 33cm
- To check phorias

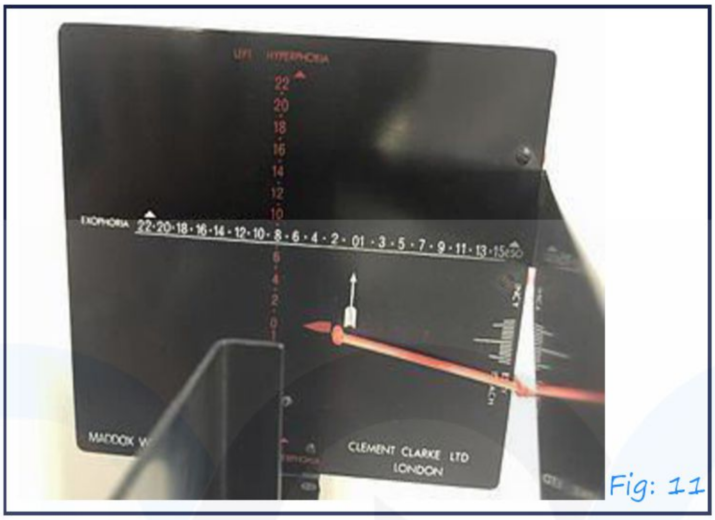

LEFT HYPERPHORIA
EXOPHORIA
22·20·18·16·14·12·10·8·6·4·2·01·3·5·7·9·11·13·15
MADDOX W
CLEMENT CLARKE LTD
LONDON

Fig: 11

- Right eye only sees white & red arrows
- Left eye sees only numbered line
- In normal individuals' arrows point to 'O'

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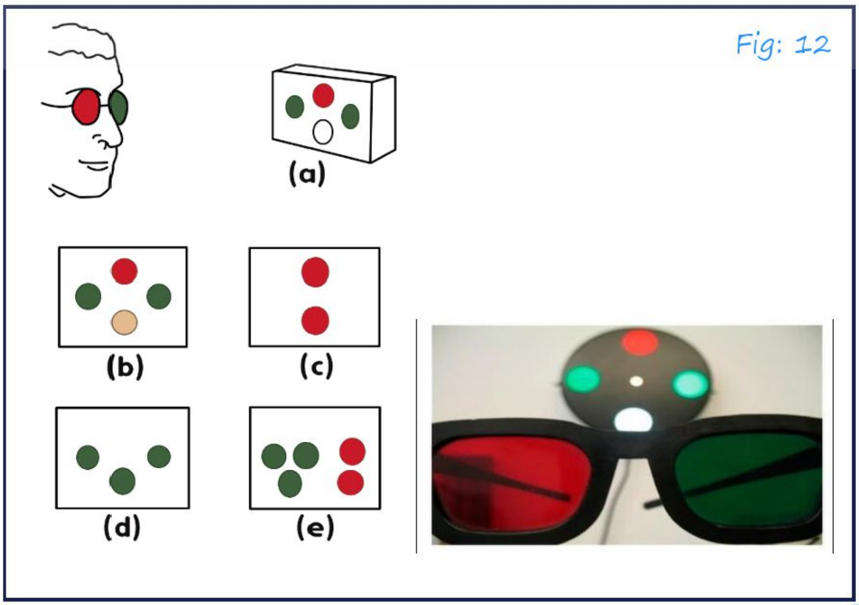
Worth four dot test 00:25:26

- It is done to check binocularity

Fig: 12



- Orthophoric – 1red, 2 green, 1 composite colour
- 2red – left suppression
- 3green – right suppression
- 3 green, 2red – Dipolopia

Stereopsis 00:28:58

- 2 pen test

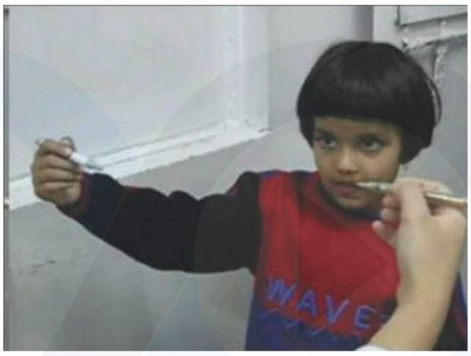


Fig: 13

- Tritmus fly test

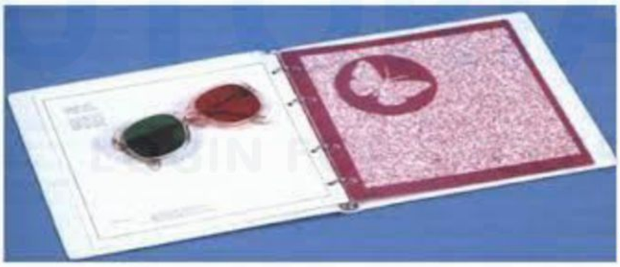


Fig: 14

- It is based on polaroid vectograph

Synaptophore

Fig: 15

Able to measure

- Grades of binocularity
- Fusion reserve
- Heterophoria
- actual amount of squint
- Orthoptic exercises – increase fusional capacity

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Forced duction test 00:34:13

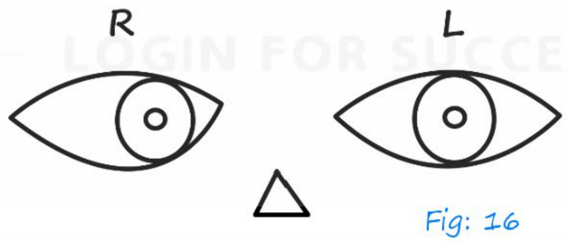

Fig: 16

- LR muscle paralysed – 6th nerve – paralytic squint or MR restricted
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- negative forced duction test → Eye is moved easily → paralysis
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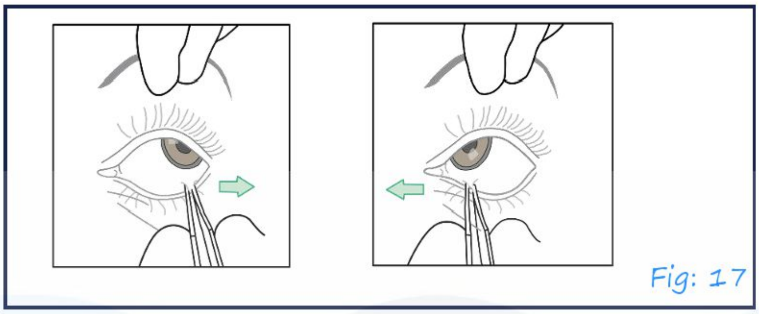

Fig: 17

Active force generation test 00:36:55

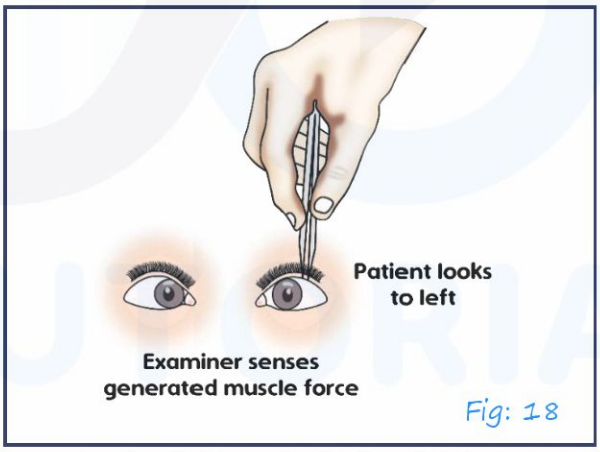
Patient looks to left
Examiner senses generated muscle force

Fig: 18

- Limbus is held with forceps
- Patient tries to move the eye

If eye does not move	Eye is able to move
↓	↓
Nothing felt by examiner	TUG is felt by examiner
↓	↓
Paralytic	Restrictive

ESOTROPIA

- Uncrossed convergent squint

Types
- Accommodative
  - Refractive
  - Non-refractive
- Non-Accommodative

Accommodative Refractive Esotropia

- High uncorrected Refractive error- Hypermetropia (+ 3D → +8D)
- Hypermetropia leads to loss of Accommodation

  ↓

  This in turn lead to overactivity of Medial rectus

  ↓

  Esotropia
- Treatment: - Convex lens to correct Hypermetropia

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Non-Refractive Accommodative Esotropia

- High Ac/A ratio → at distance: minimum esotropia

  → **at near: High esotropia

  ∴ More convergence occurs for accommodation

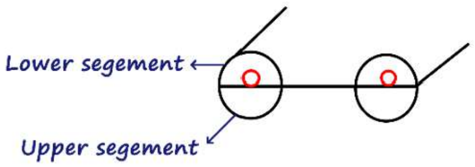
Normally

Accommodative convergence(AC) /Accommodation

Ratio(A) = 3:1

Treatment

- Since Problem is increasing in near vision – Bifocal glasses called executive Bifocals



Intersection of upper & lower segment lies at lower Border of pupil

Non-Accommodative esotropia 00:11:00

- A/K/A Essential esotropia
- Idiopathic in Nature
- No refractive error
- Ac/A ratio is Normal
- since Present at Birth/early after birth < 6 months it is also known as congenital esotropia
- High degree esotropia present
- Inferior oblique overaction is seen
- Latent Nystagmus is present
- DVD- Dissociated Vertical Deviation, (Do not follow Herring's law)
- Cross fixation present
- these children have tendency to develop Amblyopia if not corrected early

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Treatment

- Early surgery → MR Recession – Bimedially (i.e., Both eyes)
- Treat Amblyopia

Management of Squint 00:16:18

i. Retinoscopy → Based on the defect full correction is always given

ii. Orthoptics → pencil pushups → at home
→ Synaptophore – at clinic

iii. Prismatic correction – used in case of small squints by placing prism in glasses

iv. Surgery

- Recession – weakening (loosening)
- Resection – Strengthening (cut short Muscle)

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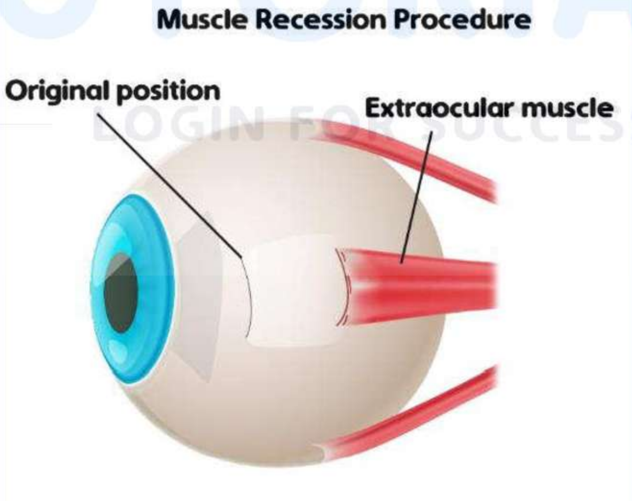
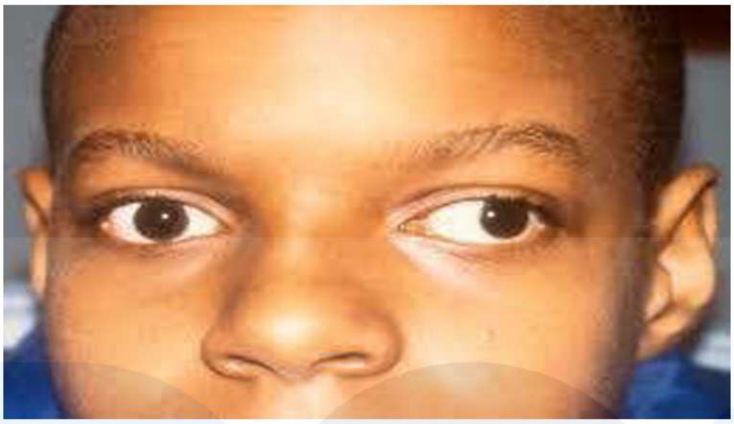

Muscle Recession Procedure
Original position
Extraocular muscle

Recession Fig: 1

- Muscle is detached from its attachment & is attached further back



Resection Fig: 2

- Leave a part of Muscle at attachment & stretch the remaining muscle and attach at original insertion
- Always a minimum of two muscles are operated

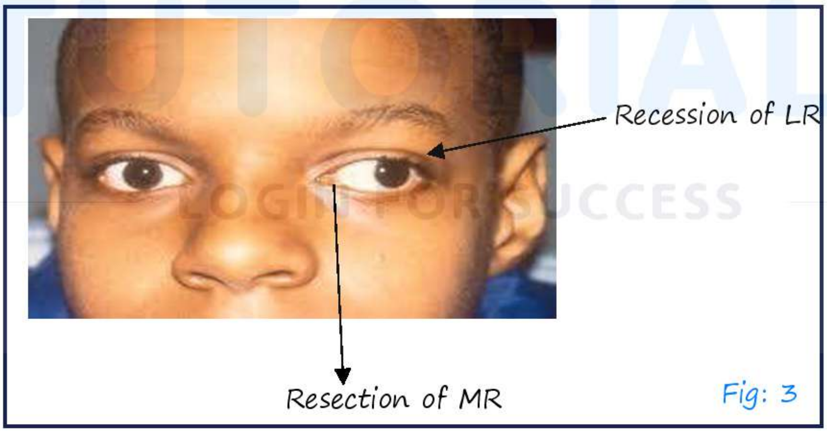

Recession of LR
Resection of MR

Fig: 3

COMITANT AND INCOMITANT SQUINT

Tropia

- Comitant squint – angle of deviation remains same in all gazes
- Incomitant squint: - angle of deviation varies in different gazes

	Comitant – congenital	**Incomitant – acquired**
Age	Early / by birth	Later in life
Vision	Poor in deviated eye	Normal
Diplopia	–	+
Abnormal head posture	–	+
Restriction of EOM	–	Paralytic / restrictive +
False projection	–	+
Primary and secondary deviation	Equal	Unequal (secondary deviation > primary deviation)

Primary and Secondary Deviation 00:05:10

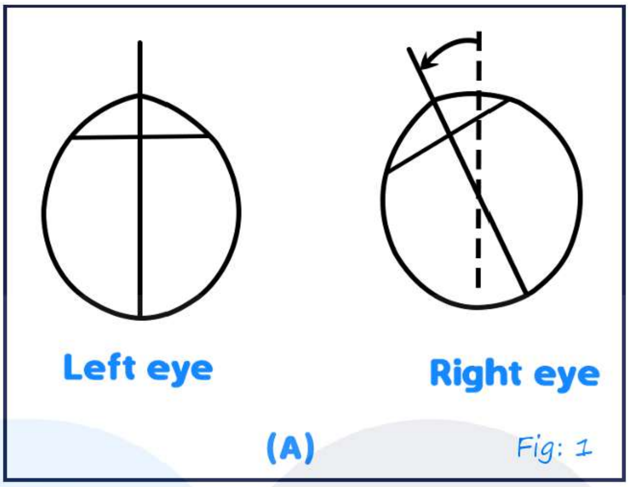
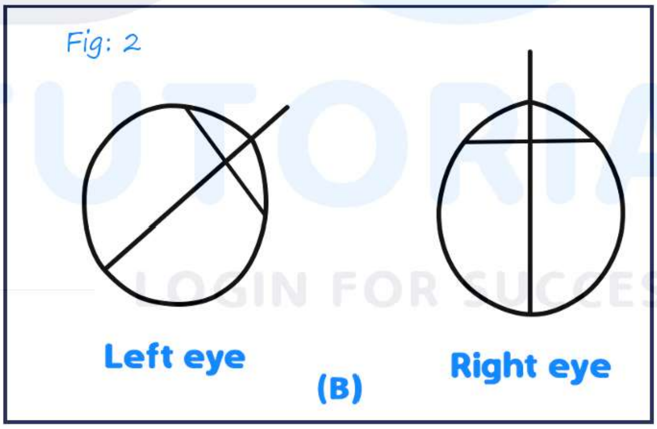

Left eye
Right eye
(A)

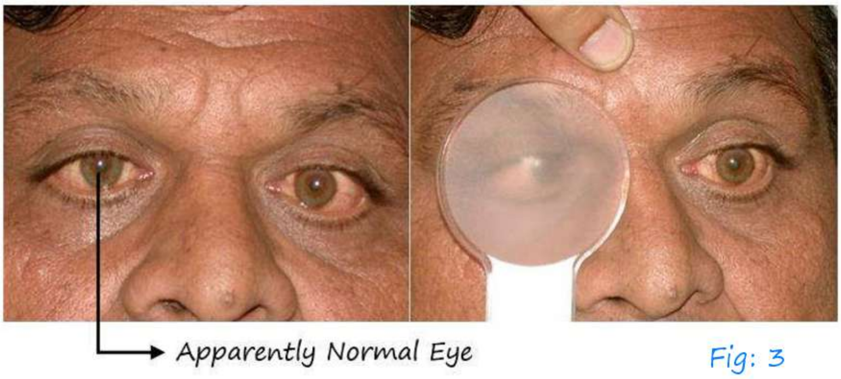
Fig: 1

A. Left eye "good eye" is fixing and there is inward deviation of the right paretic eye; (primary deviation)

Fig: 2


Left eye
(B)
Right eye

B.Right eye "paretic eye" is forced to fix and there is inward deviation of the normal left eye; (secondary deviation)


Apparently Normal Eye
Fig: 3

Primary deviation Secondary deviation

- Secondary deviation is more than primary because herring's law of equal innervation.

3RD NERVE PALSY

- All extra ocular muscles are supplied by 3rd nerve except
- – SO_4 and LR_6

Nuclei of 3rd nerve 00:01:00

– It is situated in mid brain

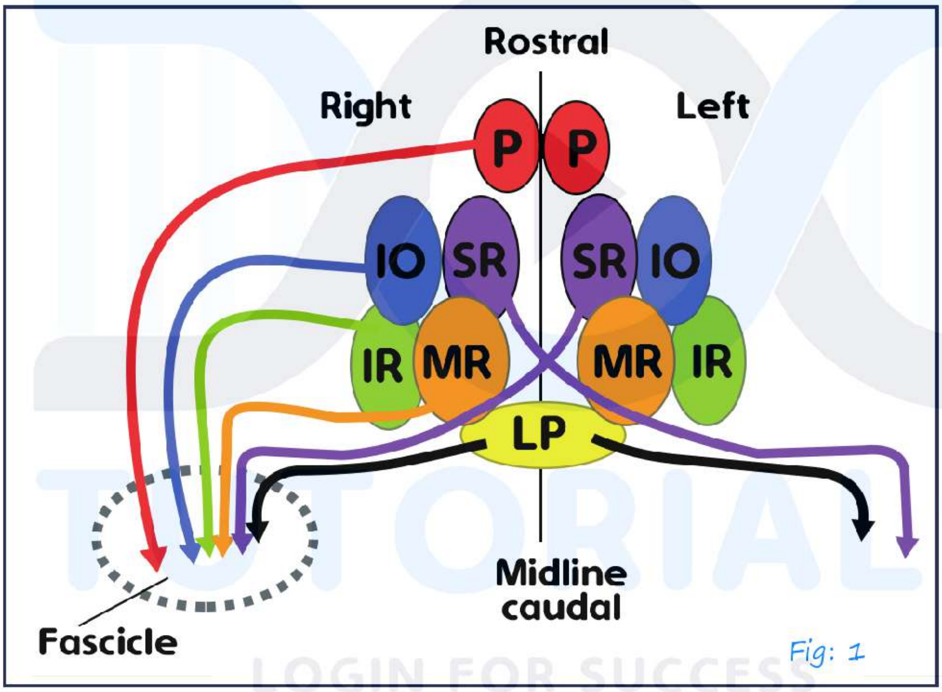

Rostral
Right
Left
P
P
IO
SR
SR
IO
IR
MR
MR
IR
LP
Midline
caudal
Fascicle

Fig: 1

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One main nucleus

Paired	Unpaired
• SR → C/L	• LPS – Midline caudal nucleus
• IR ⎤	
• MR ⎬ I/L	
• IO ⎦	

- SR –
  Innervates
  opposite side

- Apart from these motor nucleus there is a parasympathetic nucleus – EWN – Ciliary muscle and Sphincter papillae

Course of 3rd nerve: – 00:02:50

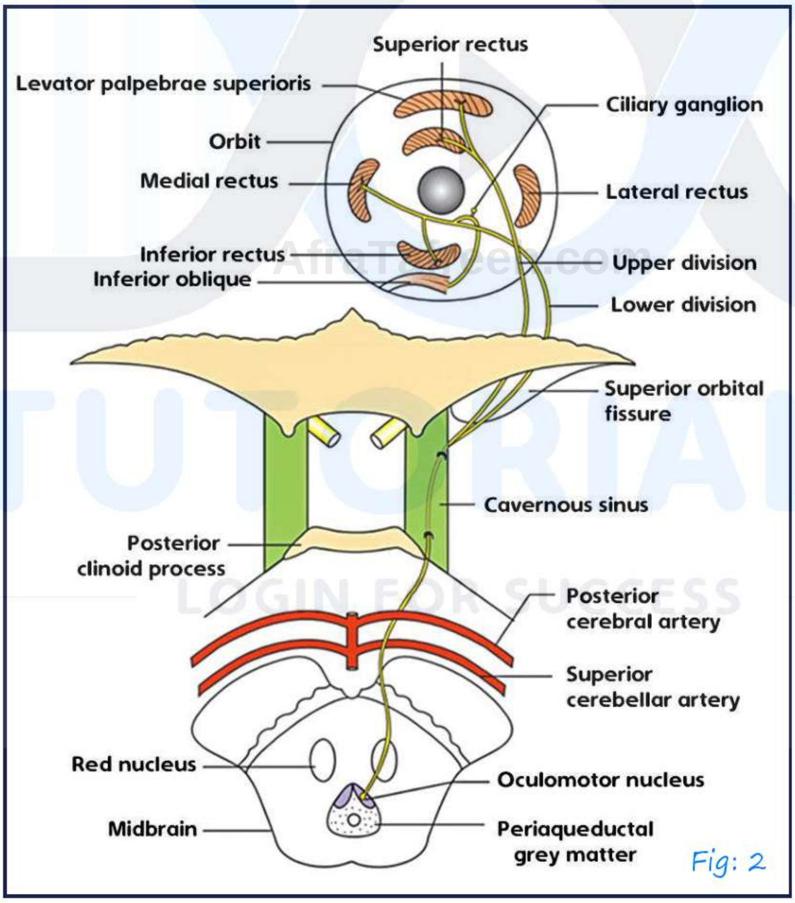

Superior rectus
Levator palpebrae superioris
Ciliary ganglion
Orbit
Medial rectus
Lateral rectus
Inferior rectus
Inferior oblique
Upper division
Lower division
Superior orbital fissure
Cavernous sinus
Posterior clinoid process
Posterior cerebral artery
Superior cerebellar artery
Red nucleus
Oculomotor nucleus
Midbrain
Periaqueductal grey matter

Fig: 2

- 3rd nerve nucleus is in midbrain at the level of superior colliculus
- Fascicular part – goes across red nucleus
  ↓

- Basilar part – goes between superior cerebellar, posterior cerebral

artery

↓

Goes through cavernous sinus along with 4th, 5th nerve

↓

Superior orbital fissure

Upper division	lower division
LPS & SR	Rest of muscles

Structures supplied by 3rd nerve

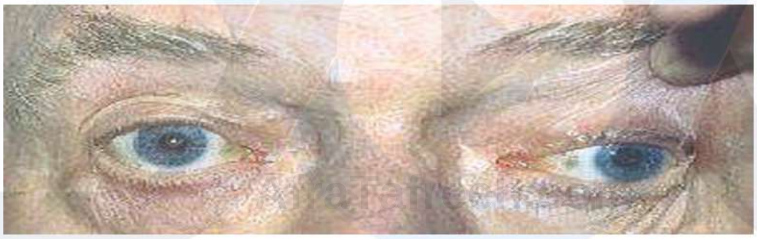
- LPS – Levator palpebrae superioris
- SR – superior rectus
- MR – medial rectus
- IO – inferior oblique
- IR – inferior rectus
- Sphincter papillae
- Ciliary muscle

Name of muscle	Normal function	If function is lost
LPS	Elevation of upper lid	PTOSIS
SR	Elevation of eye	Eye will go downward
MR	Addiction of eye	Abduction outward (LR++)

IO	Elevation of eye	Eye will go downward
Sphincter papillae	Miosis	Mydriasis
ciliary muscle	Accommodation	Loss of accommodation

- As superior oblique is intact - eye will go down
- Clinically – patient has-

Down and out eye with ptotic lid



Left Third Nerve Palsy Fig: 3

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Complaints: -

- Generally, there is no diplopia since there is ptosis but diplopia might be seen when there is mild ptosis
- Drooping of lid
- Pupil may be dilated / normal

3rd nerve palsy- pupillary sparing:

- Medical conditions → DM and HTN

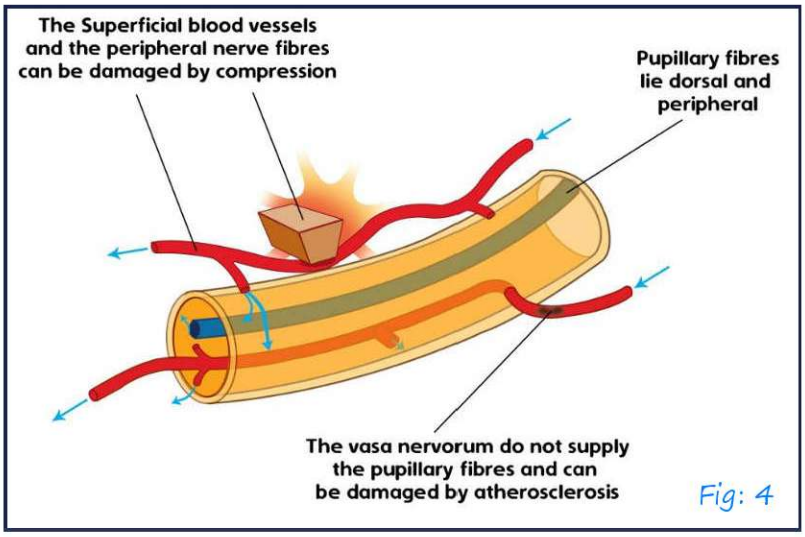

The Superficial blood vessels and the peripheral nerve fibres can be damaged by compression
Pupillary fibres lie dorsal and peripheral
The vasa nervorum do not supply the pupillary fibres and can be damaged by atherosclerosis

Fig: 4

- Nuclear lesion: there is a single nucleus for LPS. If that is compressed → bilateral ptosis
- Fascicular lesions –
  - Webers syndrome
  - Nothnagel syndrome
  - Claude syndrome
- Treatment – wait and watch; because 3rd nerve regenerates

– treat underlying conditions if present [DM, HTN]

- Some times there can be aberrant regeneration of 3rd nerve → Pseudo von graefe sign → Lid lag on looking down
- LPS is innervated by the nerve branch which also supplies IR. So, when IR contracts, LPS also contracts – when looking down

LPS + IR contracts together

Lid retraction when globe / eyeball looks down

↓

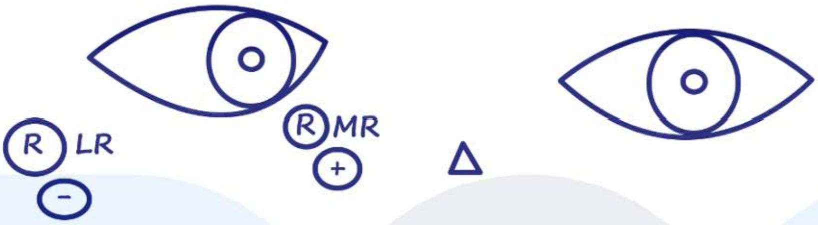
It appears as lid lag.

6TH NERVE PALSY

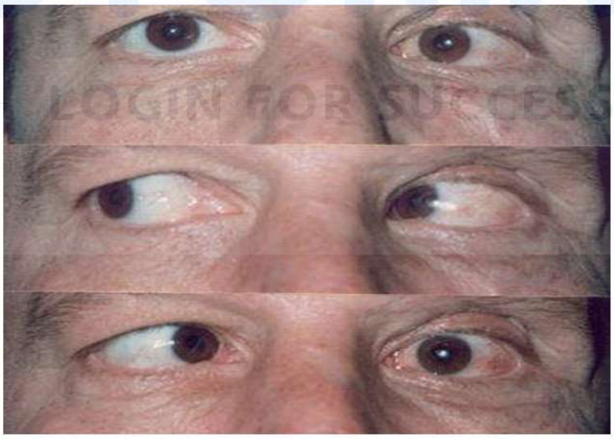
Sixth nerve palsy: -

- Lateral rectus (LR) → abduction of eye

  Eg: Right LR palsy



- Mild right esotropia in 1° gaze
- complaint of diplopia is maximum is same direction of paralysis
- To avoid diplopia- head posture → face turn on same side of paralyzed muscle.



Left LR palsy Fig: 1

4TH NERVE PALSY

- Also known as trochlear nerve, it has longest extradural course
- Only nerve to exit from dorsal aspect of mid brain

Anatomy

- Nucleus: Mid brain at level of inferior colliculus

  ↓

  Cross over occurs at dorsal aspect of mid brain

  ↓

  Basilar part of 4th C.N passes between superior cerebellar artery and
  posterior cerebral artery

  ↓

  Passes through cavernous sinus
  (Lateral wall of cavernous sinus in relation to 3rd and 5th C.N)

  ↓

  It supplies superior oblique muscle

4th C.N Palsy

- Action: Ab I D Mnemonic

  ↓ ↓ ↓

  Abduction intorsion depression
- Pure depressor in adducting eye
- This is the most common congenital palsy

Features

- Hypertropia: Due to over activity of inferior oblique
- Diplopia → vertical diplopia

  Occurs when patient looks down, (Reads / goes down stairs)
- Head posture:

  1.Chin depression

  2.Head turn to opposite side

  3.Heald tilt to opposite shoulder

Park 3 step test

1.To know which eye is hypertropic

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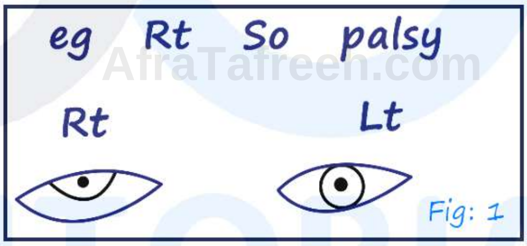

Fig: 1

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2.In which gaze hyper tropia increases

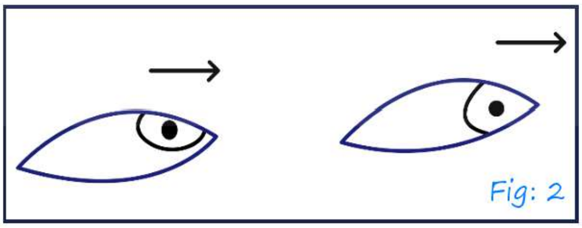

Fig: 2

Mnemonic WOOG

Worse on opposite gaze

It is due to unopposed action of inferior oblique

Therefore, head posture becomes – turning to opposite side

3.On which head tilt hypertropia improve / worsen

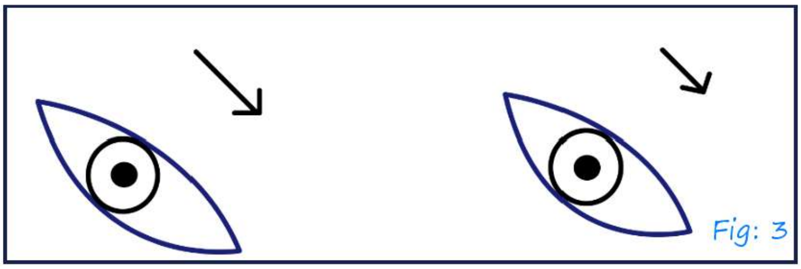

Fig: 3

Mnemonic:BOOT

Better on opposite tilt

Note

Vestibulo-ocular reflex

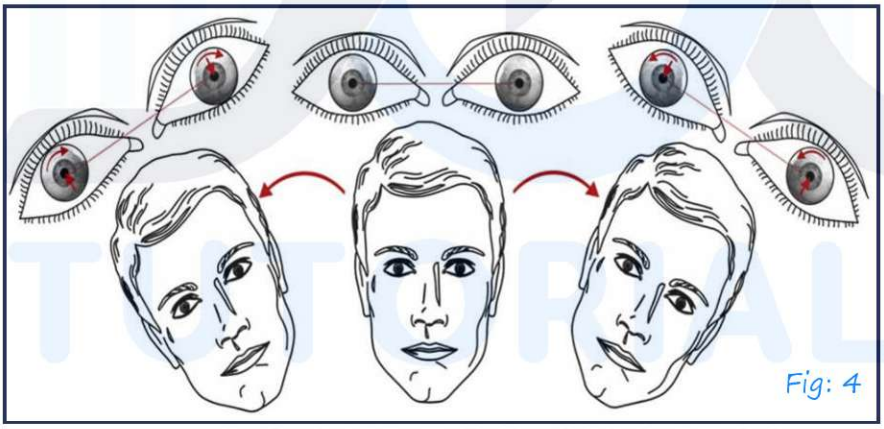

Fig: 4

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On head tilt eye on the same side undergoes intorsion and opposite eye undergoes extorsion.

Hypertropia worsen in ipsilateral head tilt:

- Intorters: Superior rectus and superior oblique
- In SO palsy, SR will compensate for weak intorsion of SO, but will elevate the eye
- And also, IO is unopposed, it again causes elevation

Thus, leading to increase in vertical separation of images and thus causing diplopia

On opposite head tilt

- The left eye has to intort and right has to extort and right eye has working IO and IR and thus no extra elevation occurs

  ↓

  Eye position is maintained and hence hypertropia decreases

Note

Beilshowsky's test: 3rd step of park 3 step test.

PATTERN SQUINT & RESTRICTIVE SQUINT

Pattern Squint

- Horizontally comitant – in horizontal gaze, Angle of Deviation remains constant
- Vertically incomitant – in vertical gaze, convergence/Divergence May occur

Patterns

- V pattern
- A pattern – Common
- X pattern
- Lambda pattern
- Y pattern

V exotropia

- Exotropia increases on up gaze
- Exotropia decreases on down page
- Most common cause – inferior oblique overaction
- If differences between up & down gaze is > 15 degrees Prism Diopters (PD) it is called V pattern

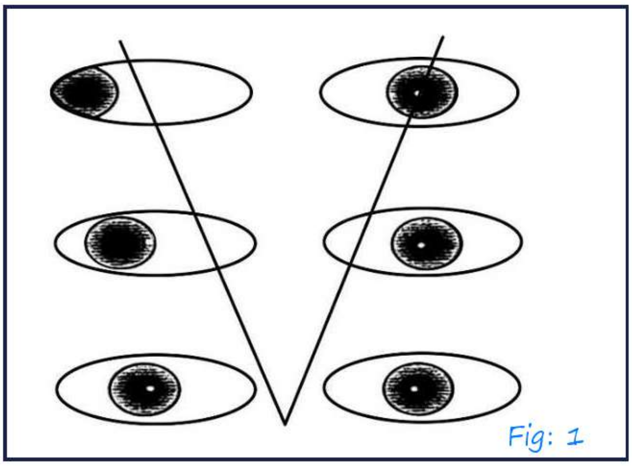

Fig: 1

A exotropia

- Exotropia increases on downgaze
- Exotropia decreases on up gaze
- Most common cause– superior oblique overactivity
- If Difference Between up & down gaze is 10 PD – it is called A pattern

---

Restrictive Squint 00:04:40

Duane's Retraction Syndrome (DRS)

- 3 Types of DRS
- **Most common type1** – limitation of abduction
  - Adduction – Normal + Retraction of globe with Narrowing of palpebral aperture seen

Cause:

- Abnormality of 6th Cranial Nerve (or) 6th cranial Nerve nucleus is present as result of this, LR is also innervated by 3rd C.N

TUTORIALS LOGIN FOR SUCCESS

↓

∴ Both MR & LR are stimulated simultaneously leading to Retraction of globe & narrowing of palpebral aperture

- **Type 2**: Limitation of adduction
- **Type 3**: Limitation of adduction + abduction

Brown's Syndrome

- Congenital click Syndrome/Congenital superior oblique Restriction

Syndrome
↓
Mimics as inferior oblique palsy,
limitation of elevation of adducted Eye

AMBLYOPIA

- Also known as lazy eye
- It is any decrease in vision which cannot be further corrected in absence of any organic cause
- Visual acuity < 6/12 → Amblyopia
  or difference of 2 lines on snellen chart
- Definition: - Any decrease of vision because of absence of stimulus / abnormal binocular interaction

Cause / Theories 00:03:55

1. Stimulus deprivation: - Eg: - Congenital cataract, ptosis since birth
   - Worst prognosis
2. Strabismic: - Large angle squint since birth
3. Anisometropic: - uncorrected high refractive error of one eye or high difference in refractive error of both eyes is 6D → Amblyopia
4. Organic → Shrinkage of lateral geniculate body
   - Amblyogenic age – Till 8 years of life
   - Amblyopia should be corrected before this age

Clinical features 00:06:40

- Crowding phenomenon
  - Individual letters are seen better as compared to lines

Treatment 00:07:58

- Treat as soon as possible

  1.Occlusion of normal eye → TOC

- Occlusion / patching

Fig: 1

Occlusion

Normal eye : Amblyopic eye

1:1 – if age is 1 year

2:1 – if age is 2 years

And so on …. till 6 years

6:1 – even above 6 years

Full day / partial occlusion can be also done.

2.Penalization: - Keeping normal eye on atropine

3.CAM stimulators

MECHANICAL INJURIES

Ocular Trauma 00:00:30

Mechanical injury   Chemical injury

Mechanical Injuries 00:00:45

- BETT's classification – Birmingham Eye Trauma Technology

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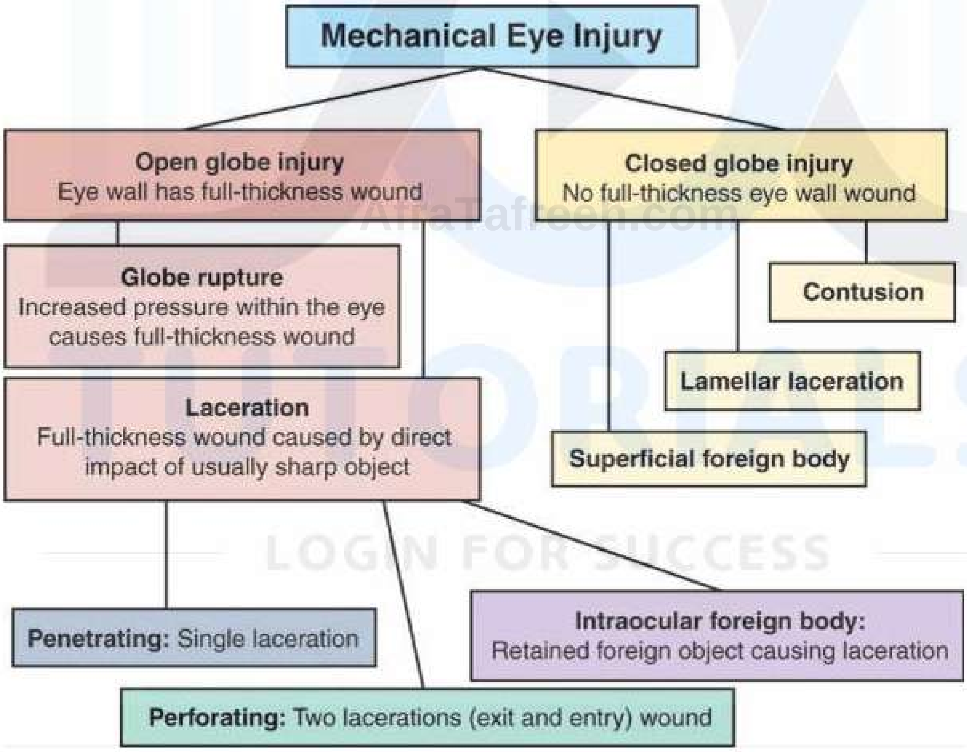
Mechanical Eye Injury
Open globe injury
Eye wall has full-thickness wound
Closed globe injury
No full-thickness eye wall wound
Globe rupture
Increased pressure within the eye causes full-thickness wound
Contusion
Lamellar laceration
Laceration
Full-thickness wound caused by direct impact of usually sharp object
Superficial foreign body
Penetrating: Single laceration
Intraocular foreign body: Retained foreign object causing laceration
Perforating: Two lacerations (exit and entry) wound

Globe Rupture 00:03:45

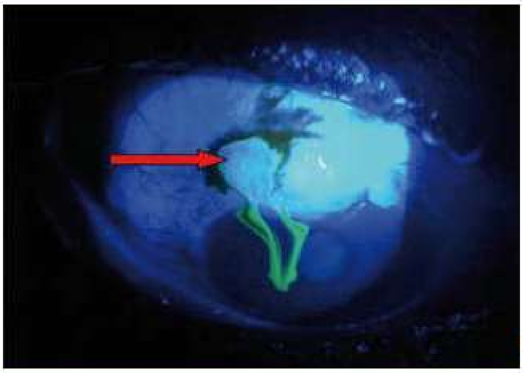

Fig: 1

- Mechanism – inside out rupture
- Most common site - super Nasal sclera – near the limbus
- signs – Hypotony

Seidel Test

- If it is +ve that means there is a leak and rupture

Eyelid Manifestations 00:06:45

- Ecchymosis –

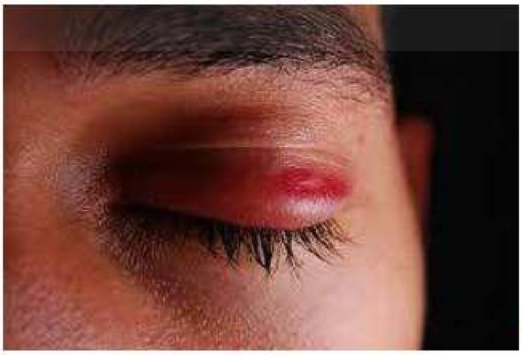

Fig: 2

- Laceration of lid
- Panda/Racoon eye

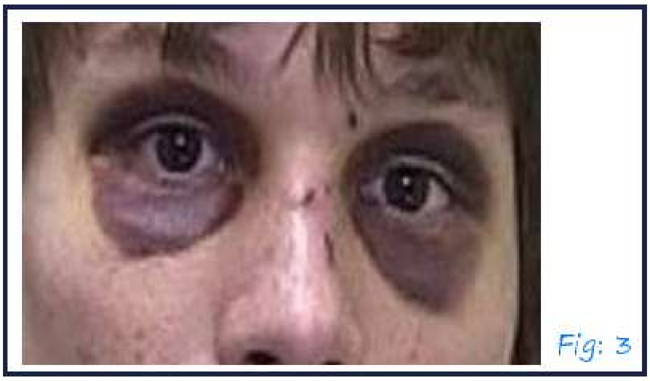
Fig: 3

- Here the trauma is at roof of orbit or anterior cranial fossa

Blowout fracture 00:07:40

- When we have a blunt injury to orbit
- It is the fracture of floor of orbit
- Types Pure – Involving orbital bones only (floor)
  Impure – Involving floor + Rim

Clinical Features

- Diplopia – double diplopia
  ↓
  Diplopia on looking both up and down
  - It is because of entrapment of IR within the fractured segment
- Loss of sensation on ½ half of the face
  - it is because of Injury to Infra orbital Nerve
- Enophthalmos – in late stage
  - It is sunken eye

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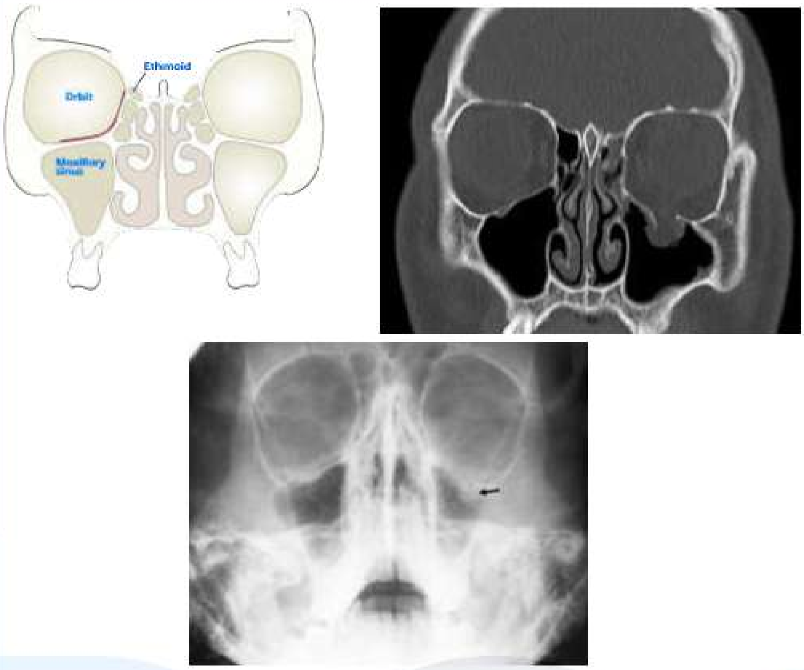

Tear drop sign Fig: 4

- Here the maxillary sinus gets contents from orbit that is why there is sunken eye

Forced duction Test: –

- If it is +ve it means there is restriction of IR muscle

Indication of Treatment

- If enophthalmos > 2mm
- If diplopia is persisting even in 1° gazes
- If fracture segment is more than ½ of length of floor

Canalicular Injury 00:14:35

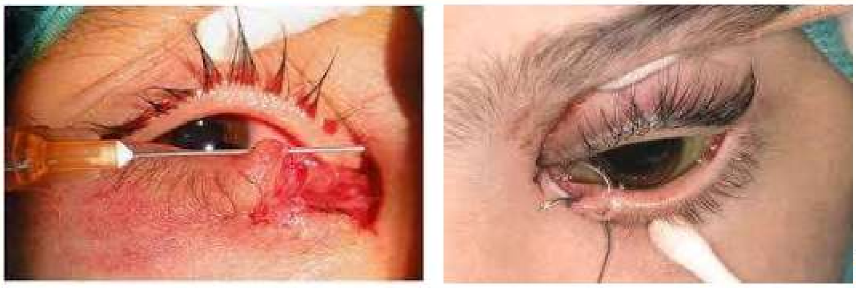

Fig: 5

- It is repaired properly after placing stent

Conjunctiva Manifestations 00:15:27

- Laceration
- sub conjunctival hemorrhage (it is most common in blunt trauma)
- It is self-resolving (2-3weeks) - usually supplements are given
- If posterior limit of hemorrhage is not seen then we have to find the extension

Corneal Manifestations 00:16:50

- Corneal epithelial defect
- laceration/perforation
- corneal blood staining

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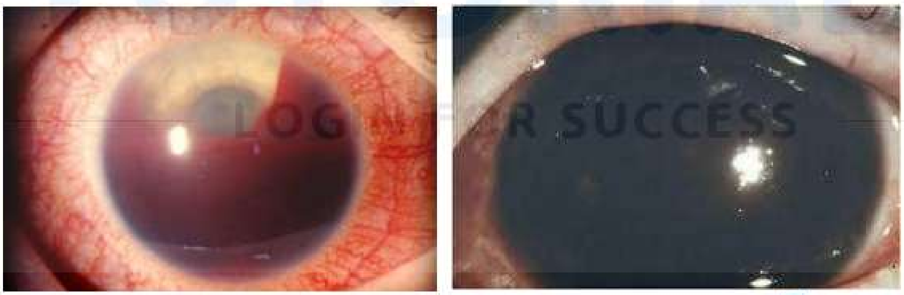

Fig: 6

- Hyphemia- Blood in anterior chamber

  ↓

  On long standing -corneal staining is seen

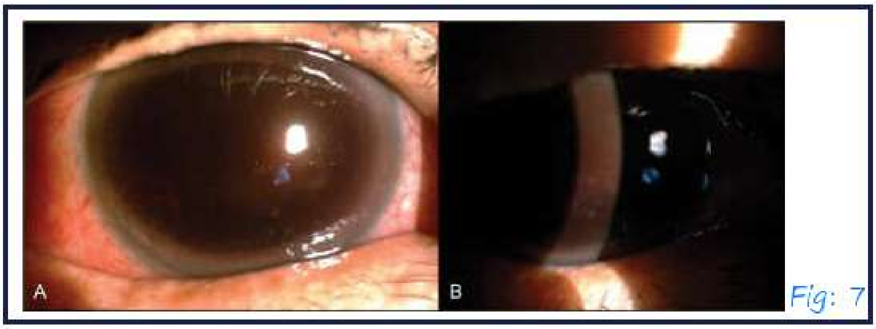

Fig: 7

- This corneal blood staining cause decrease in vision, takes years to resolved
- This corneal staining finally becomes black color resembling 8 ball and this is due to methemoglobin
- IOP may rise and results in Glaucoma it is treated with rest and topical steroids

  - It IOP > 60 mm for 32 das
  - IOP >25 mm Hg for 5 days

  Surgical intervention is done

Lens manifestations 00:18:20

- Early Rosette Cataract: Feathery lines of opacities along the star-shaped suture lines in posterior cortex
- Late rosette cataract : Posterior cortex, 1 to 2 years after the injury.
- Its sutural extensions are shorter and more compact

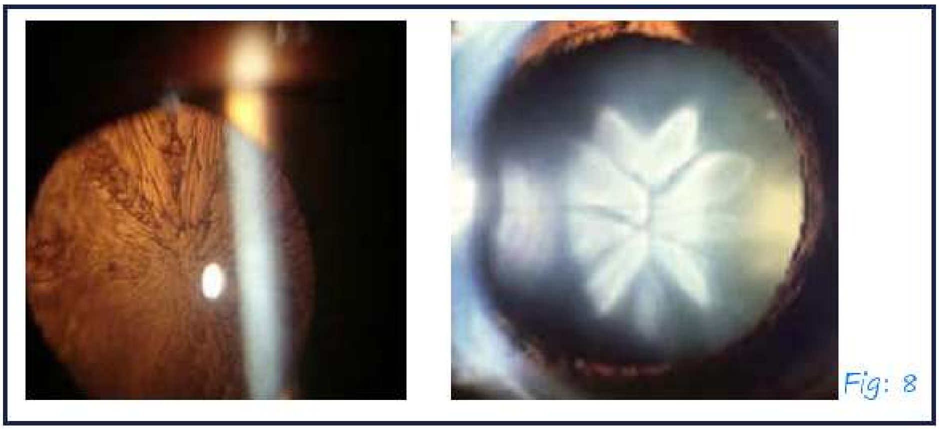

Fig: 8

- We can also have subluxation / dislocation of lens

Vossius ring 00:18:30

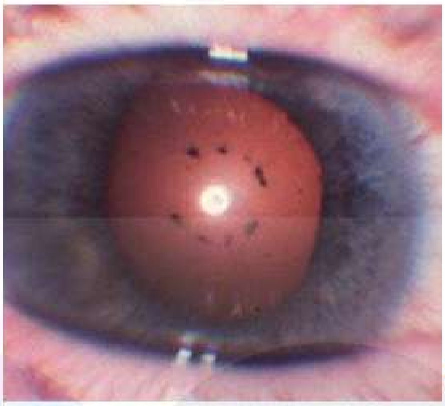

Fig: 9

- It is seen on anterior capsule of lens

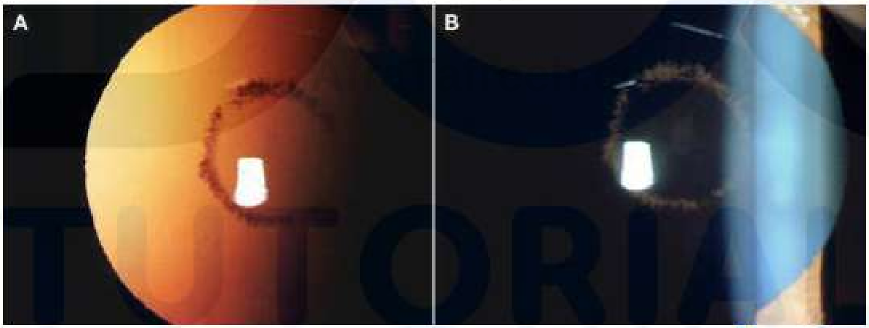

Fig: 10

- This is seen as a result of blunt trauma causing compression of Iris over lens and the iris remnants which resemble this ring
- This ring is smaller than the size of pupil
- It doesn't disturb vision
- It is Reversed

IRIS manifestation 00:22:05

- Traumatic meiosis and Mydriasis

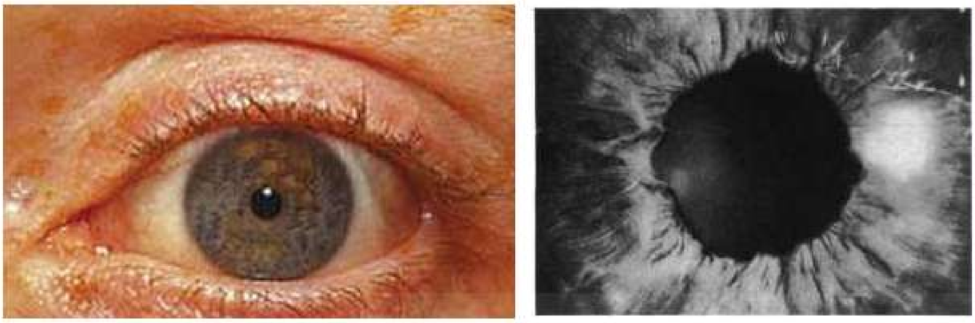

Fig: 11

Iridodialysis 00:22:40

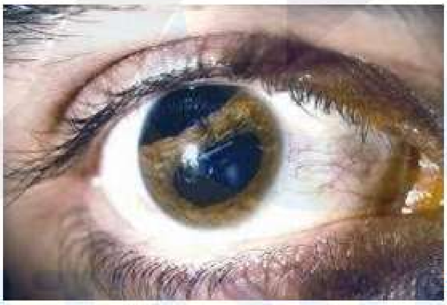

Fig: 12

- Because of blunt trauma IRIS gets separated from the root
- There is a D shape pupil which results in Mono-ocular diplopia
- Anteflexion of Iris

Early Rosetter Cataract

- Feathery lines of opacities along the star shaped suture lines in posterior cortex

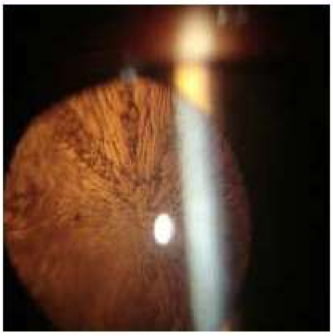

Fig: 13

Late Rosetter Cataract

- Posterior cortex, 1 to 2 years after the injury.
- Its sutural extensions are shorter and more compact.

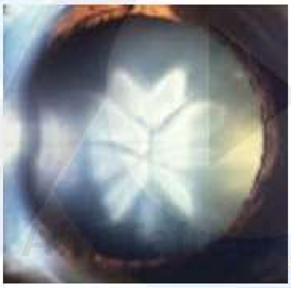

Fig: 14

- Angle recession glaucoma-irregular widening of ciliary body band

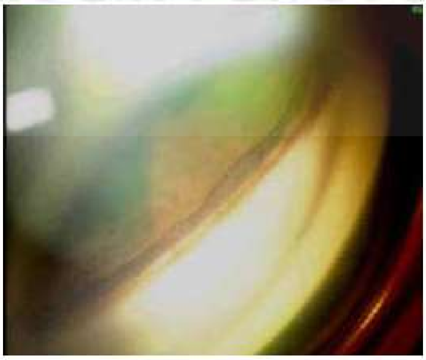

Fig: 15

- It is seen in 4-5 of patients
- It is seen in Gonioscopy – there is separation of longitudinal & circular muscle of ciliary body

- If there is a patient with increase IOP – Do gonioscopy
  ↓
  unexplained high IOP
- If there is angle recession on gonioscopy
- There is 40% chance of developing glaucoma

Posterior segment manifestations 00:26:45

- Seen in vitreous, Retina, choroid
- 1. vitreous hemorrhage
  - Long standing vitreous hemorrhage results in 'ghost cell glaucoma'
- 2. Commotio retinae
- 3. Retinal tear /Rhegmatogenous RD
- 4. Retinal dialysis
- 5. Vitreous Base avulsion
- 6. Choroidal rupture

Commotio Retina Vs Berlins Edema 00:28:20

- Here the Retinal whitening is not because of edema

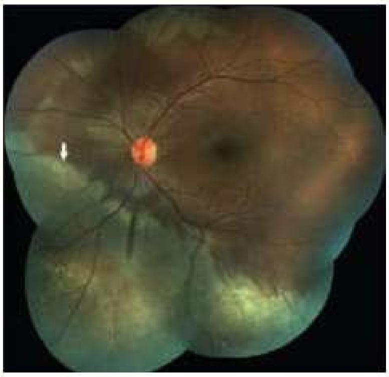

Fig: 16

- Characteristic glistening appearance described below is likely related to increased scattering of light by the abnormal photoreceptor processes

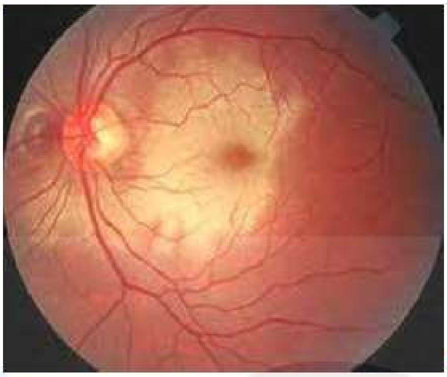

Fig: 17

- This whitening is indicative of cell damage, which occurs in the RPE and outer segment layer of photoreceptors.
- Results in photoreceptor death.
- Usually there is no leakage of fluid and therefore it is not considered a true edema
- Berlin edema is seen in posterior pole
- Characteristically we see cherry red spot
- This is a self-limiting condition, usually gets bk to normal within a month

Choroidal Rupture 00:30:08

- The retina is elastic and stretches during injury.
- Bruch membrane breaks because it does not have sufficient elasticity.

- The choriocapillaris is injured and bleeds into the sub RPE and/or subretinal space
- Crescent shaped yellowish subretinal streak concentric with the optic disc.

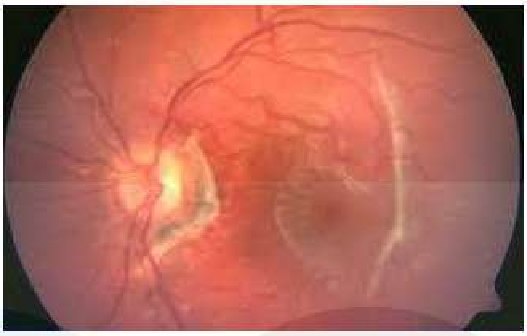

Fig: 18

Retinal dialysis 00:30:37

- IT most common
- But with trauma SN mc
- Tear in the retina whose anterior edge is at the ora serrata and whose posterior edge is attached to the vitreous base

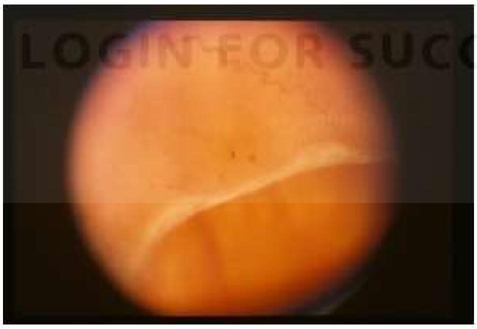

Fig: 19

- Inferotemporal Retinal dialysis is most commonly seen but with trauma it is super nasal which is common

Vitreous base evulsion 00:31:15

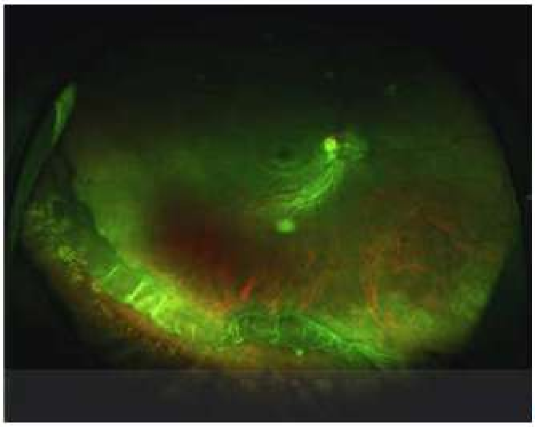

Fig: 20

- Pathognomic sign of blunt trauma

Signs at level of Optic Nerve 00:31:50

- 1° optic atrophy is seen
- Intracanalicular part – most common damaged it is near the junction of lateral 1/3rd medial 2/3rd
- optic nerve evulsion may see rarely

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7 rings of trauma 00:32:50

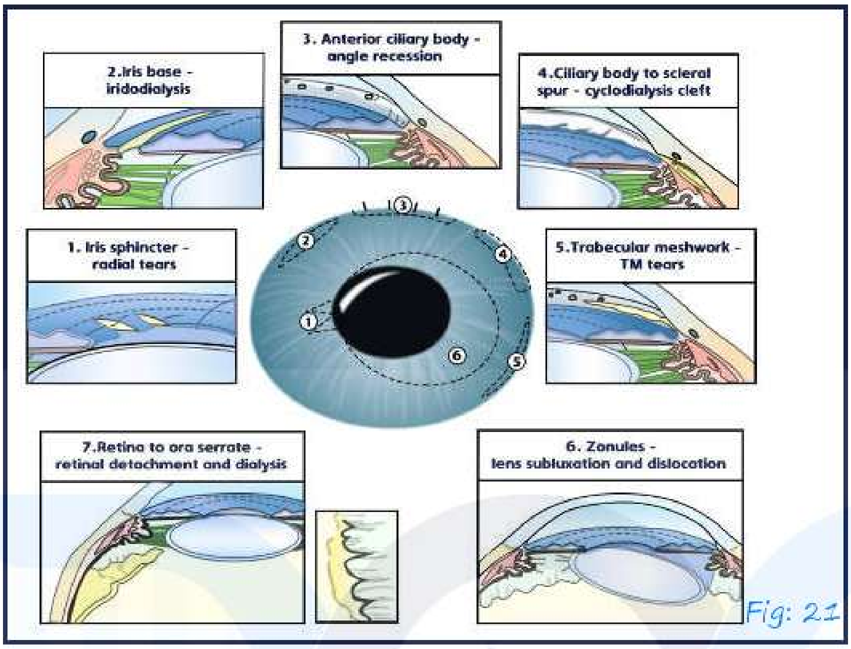
2.Iris base - iridodialysis
3. Anterior ciliary body - angle recession
4.Ciliary body to scleral spur - cyclodialysis cleft
1. Iris sphincter - radial tears
5.Trabecular meshwork - TM tears
①
②
③
④
⑤
⑥
7.Retina to ora serrate - retinal detachment and dialysis
6. Zonules - lens subluxation and dislocation

Fig: 21

Penetrating Injuries 00:34:00

- Most common Intraocular foreign body – Iron & chisels toxic & insert materials

Toxic	Inert
• Metallic – Magnetic -iron, steel, nickel – Non-magnetic – Copper, aluminum, mercury, zinc	• Metallic -Gold, silver, platinum
• Non- Metallic – vegetative matter	• Non-metallic – Glass carbon stone, porcelain, plaster, rubber

TUTORIALS LOGIN FOR SUCCESS

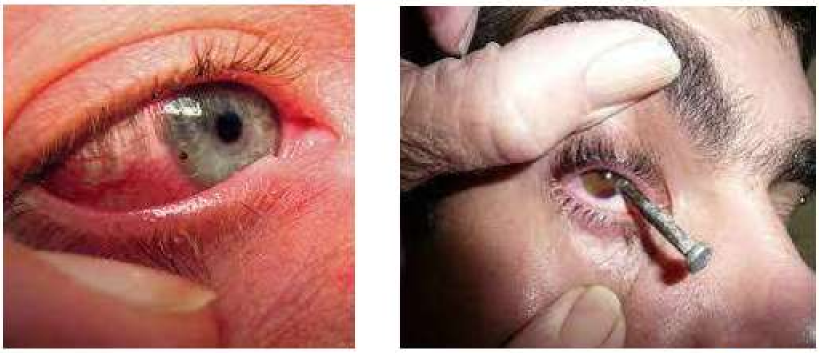

Fig: 22

Siderosis Bulbi 00:36:50

- Intraocular accumulation of Iron
- Degenerative changes produced by an iron foreign body
- 2 months to 2 years of the injury
- MECHANISM – Iron particle undergo electrolytic dissociation and disseminated throughout the eye.
- These ions combine with intracellular protein and produce degenerative changes mainly in epithelium

Signs of siderosis bulbi

- In anterior lens capsule
- Hyperchromic iris
- Secondary glaucoma
- Pigmentary retinal degeneration
- ERG - Decrease in b wave of ERG is seen which may later become flat

TUTORIALS LOGIN FOR SUCCESS

Chalcosis 00:38:40

- Specific changes produced by alloys of copper in the eye
- IOFB with high copper content → violent endophthalmitis
  → phthisis bulbi
- Alloys with low copper content brass/ bronze
  →chalcosis

Chalcosis

- Deposition on membranous structures
- KF ring
- Sunflower cataract

DOT TUTORIALS LOGIN FOR SUCCESS

Localization of IOFB 00:40:00

- X-Ray Antero-posterior and lateral views as most foreign bodies are opaque.
- Limbal Ring Method
  - Metallic ring of corneal diameter stitch at limbus and x ray in 3 postures patient looking straight, up and down

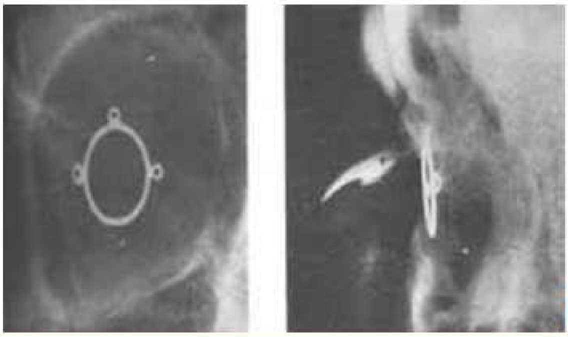

Fig: 21

- MRI is absolutely contraindicated if the foreign body is Iron, we can go for Ct, ultrasound B scan
- USG B scan showing IOFB- hyperechoic, shadowing'

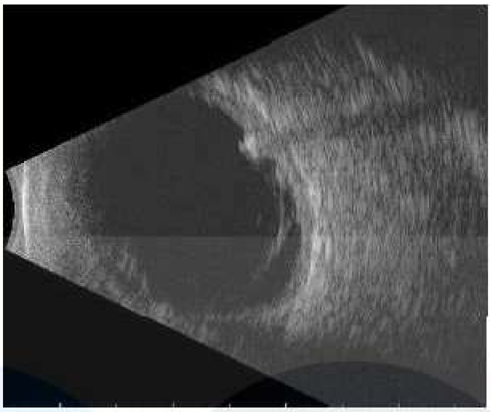

Fig: 22

Sympathetic ophthalmia 00:41:10

- It is B/L pan uveitis granulomatous, non-suppurative after penetrating injury → especially after ciliary boy injury

  ↓

  Danger area of eye
- Traumatic eye- inciting eye
- The other eye- sympathizing eye/non traumatic eye
- It can occur after 2 weeks or many years
- 1st symptoms → loss of accommodating (difficulty in reading)
- 1st sing: Retrolental flare

  ↓

  Behind the lens
- Dalen Fuch Nodules: collection of epitheloid cells at the level of RPE

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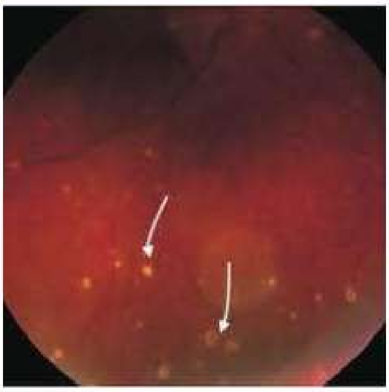


Fig: 23

Treatment

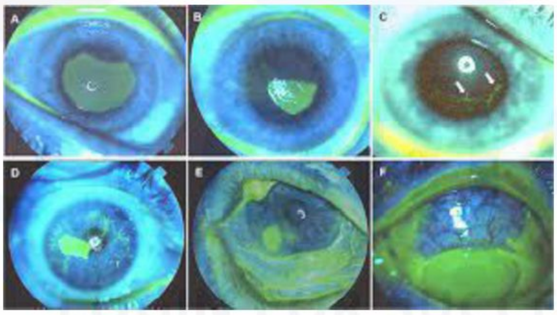
- Steroids
- Immunomodulatory drugs
- Best prophylaxis – Enucleation of the traumatic eye

CHEMICAL INJURIES

Chemical Injuries

Acid Injuries
↓
Coagulates on surface
↓
Doesn't go much deeper
↓
Except – Hydrofluoric acid
↓
Can go deeper

Alkali Injuries
↓
More dangerous to eye
↓
Deeper
↓
Necrosis

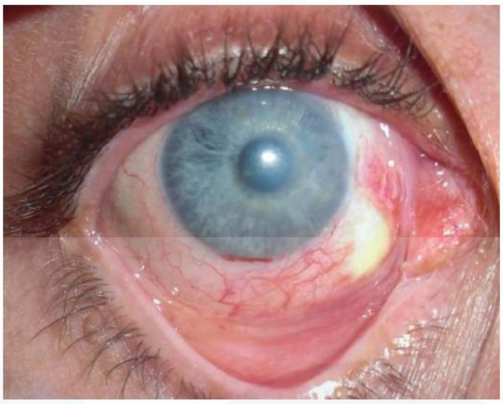


Grades of chemical Injuries to eye Fig: 1

Management 00:01:50

- Immediate management – Wash the eye
  ↓
  For 30 minutes
  or
  Till pH- 7.2

  - Ideal fluid to wash – Ringer lactate (ideal) > tap water
- Early Management: - Refer to a ophthalmologist



Limbal Ischemia Fig: 2

Roper Hall Grading 00:03:50

Grade	Prognosis	Lamble ischemia	Corneal involvement
I	Good	None	Epithelial Danage
II	Good	Less than 1/3	Corneal haze, iris details visible
III	Guarded	1/3 to ½	Total epithelial loss, stromal haze, iris, details obscured
IV	Poor	Over ½	Corneal plaque, iris and pupil are obscured

TUTORIALS LOGIN FOR SUCCESS

DUA's classification 00:04:23

Grade	Prognosis	Clinical findings	Conjunctival involvement
I	Very good	0 clock hours of limbal involvement	0%
II	Good	≤3 clock hours of limbal involvement	≤ 30%
III	Good	3-6 clock hours of limbal involvement	30-50 %
IV	Good – guarded	6-9 clock hours of limbal involvement	50-75%
V	Guarded-Poor	9- <12 clock hours of limbal involvement	75-<100 %
VI	Very Poor	Total (12 clock hours) limbal involvement t	100%

DX TUTORIALS LOGIC FOR SUCCESS

Treatment in Early Management

- Tear supplements
- Topical steroid → decrease the inflammation

  → only for one week

  ↓

  If given for more days there may be corneal melting
- NSAID's
- Tab vit-C 1 gm orally → helps in collagen synthesis
- Vitamin- A
- To lower IOP → antiglaucoma agents
- Cycloplegic drugs
- Tetracyclines → decrease Matrix Metalloproteinases
- Sodium citrate] decrease proteolytic activity

  Sodium Ascorbate

Late Management

- Limbal stem cell Transplantation
- Amniotic membrane transplant
- Keratoprosthetics
- PK- penetrating keratoplasty

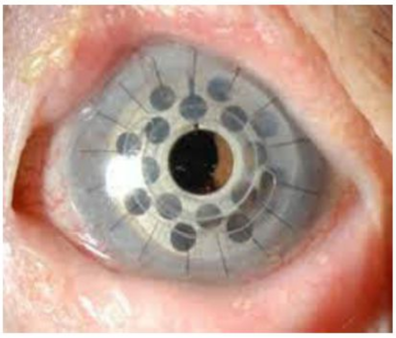


Fig: 3

ANATOMY OF ORBIT

Occuloplasty

- It deals with problem of adenexa i.e., orbit, eyelid, lacrimal apparatus

Anatomy of orbit: 00:00:23

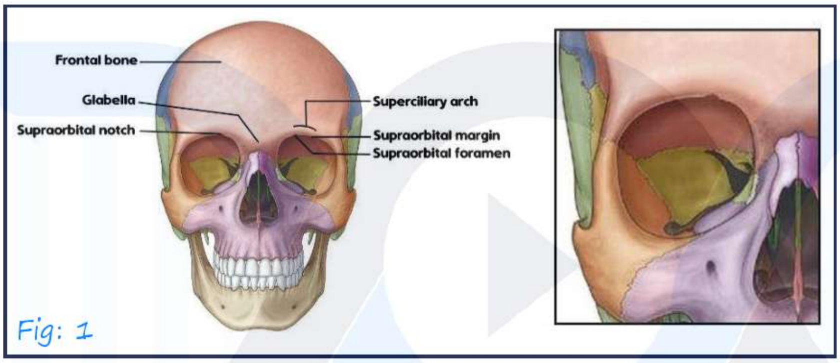

Frontal bone
Glabella
Supraorbital notch
Superciliary arch
Supraorbital margin
Supraorbital foramen

Fig: 1

- Volume of bony orbit → 30 ml

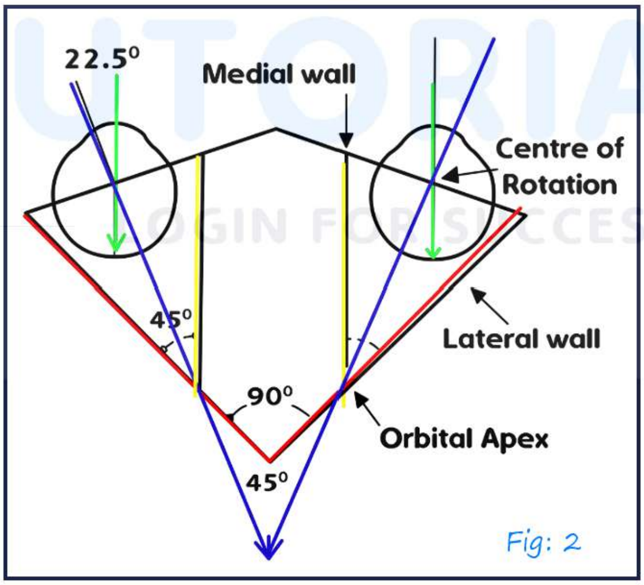

22.5°
Medial wall
Centre of Rotation
45°
Lateral wall
90°
Orbital Apex
45°

Fig: 2

- Medial walls of orbit are parallel to each other
- Lateral walls of orbit are perpendicular to each other
- Orbit is more divergent in newborns

Various distances

- ICD – Inter Canthal distance – it is the distance between two medial canthus
- IPD – Inter Pupillary distance – it is the distance between central reflection coming from two pupils
- OCD – outer Canthal distance – it is the distance between two outer canthus

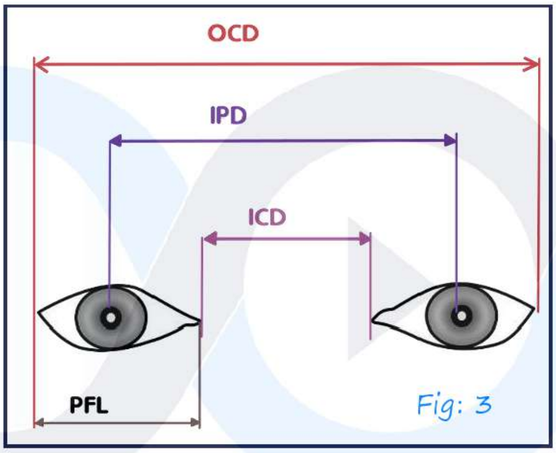

OCD
IPD
ICD
PFL

Fig: 3

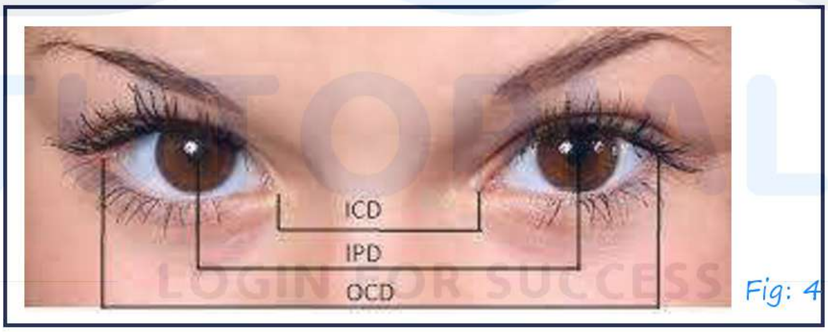


Fig: 4

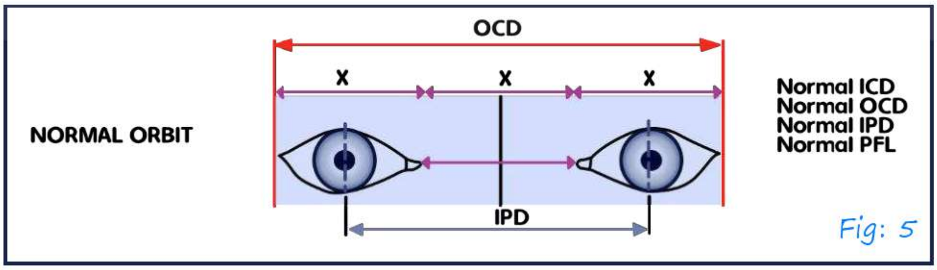

OCD
X
X
X
NORMAL ORBIT
IPD
Normal ICD
Normal OCD
Normal IPD
Normal PFL

Fig: 5

Telecanthus 00:03:24

- It is a soft tissue problem
- Long medial canthal tendons → ICD increased

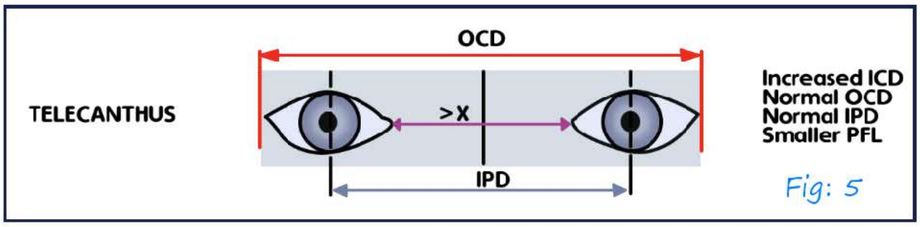

OCD
TELECANTHUS
>X
IPD
Increased ICD
Normal OCD
Normal IPD
Smaller PFL

Fig: 5

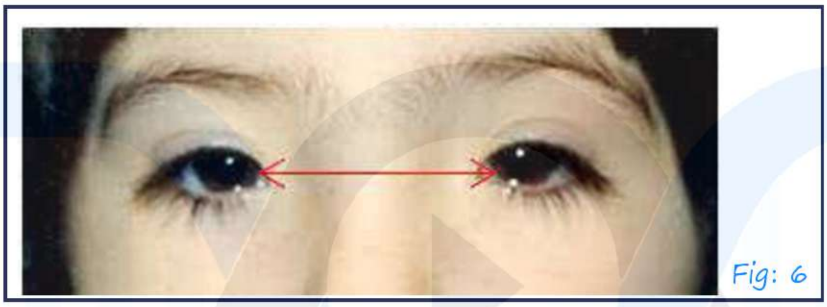


Fig: 6

Orbital Hypertelorism 00:03:24

- Widely spaced orbit
- It is a bony problem

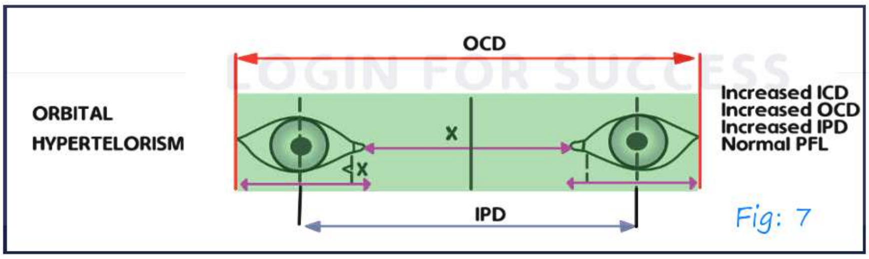

OCD
ORBITAL HYPERTELORISM
X
X
IPD
Increased ICD
Increased OCD
Increased IPD
Normal PFL

Fig: 7

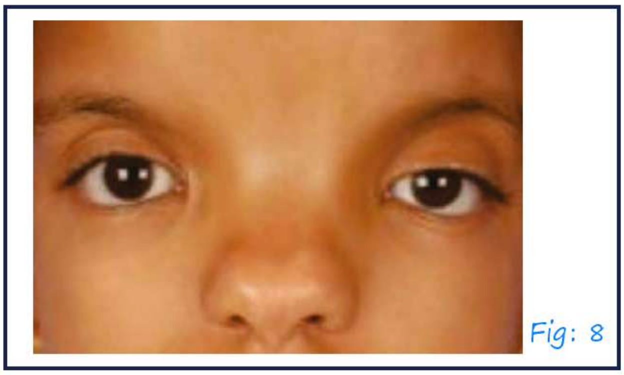
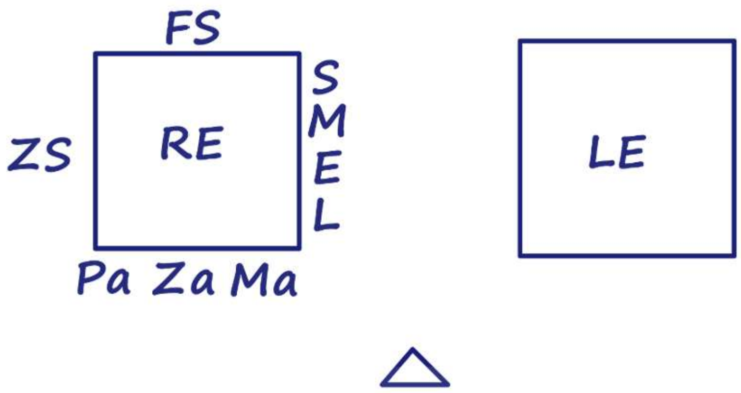


Fig: 8

Walls of Orbit 00:03:24



- Medial wall is formed by – Mnemonic – SMEL

  S- sphenoid

  M- Maxillary

  E- Ethmoid

  L- Lacrimal

- From anterior to Posterior- Mnemonic – MLE- step

  M- Maxilla- frontal process

  L- Lacrimal bone

  E- Ethmoid bone- orbital plate

  S- sphenoid bone- Body

- Weakest wall of orbit – Medial wall
- Inferior wall formed by – Mnemonic Pa Za Ma

  Pa- Palatine

  Za- Zygomatic

  Ma- Maxillary

- Sphenoid bone-

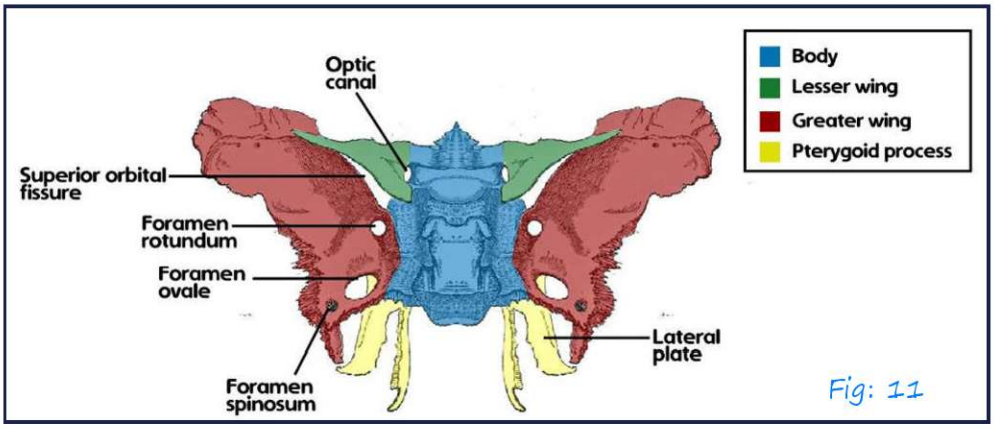

Optic canal
Superior orbital fissure
Foramen rotundum
Foramen ovale
Foramen spinosum
Lateral plate
🟦 Body
🟩 Lesser wing
🟥 Greater wing
🟨 Pterygoid process

Fig: 11

- Floor of the orbit – liable to get fractured when there is a blow → Blow out fracture seen in blunt trauma
- Lateral wall – Strongest of all
- Orbit is a- quadrangular pyramid

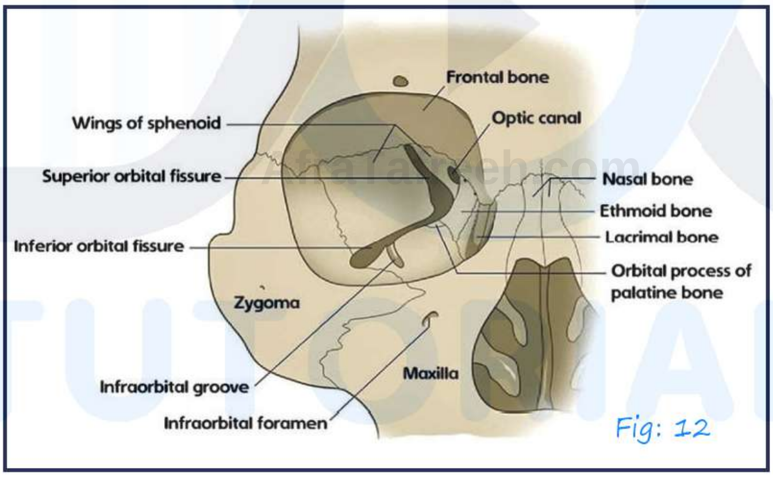

Frontal bone
Wings of sphenoid
Optic canal
Superior orbital fissure
Nasal bone
Ethmoid bone
Lacrimal bone
Inferior orbital fissure
Orbital process of palatine bone
Zygoma
Maxilla
Infraorbital groove
Infraorbital foramen

Fig: 12

- Superior orbital fissure – in between roof and lateral wall of orbit; between two wings of sphenoid
- Optic canal → lies superomedially between junction of Roof, medial wall of orbit; between body, lesser wing of sphenoid

Orbital apex anatomy

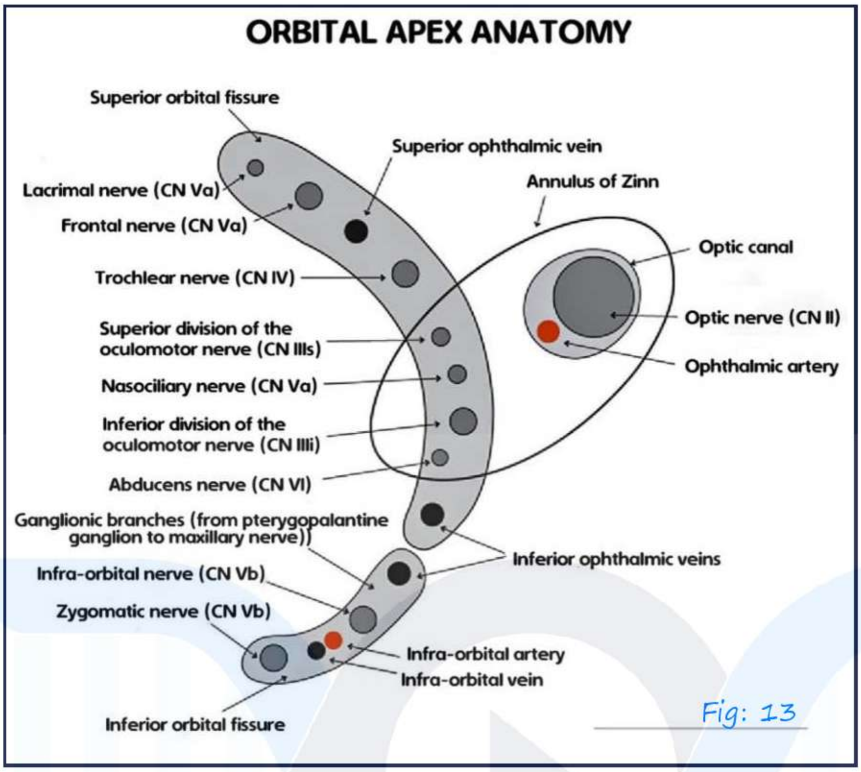

ORBITAL APEX ANATOMY
Superior orbital fissure
Superior ophthalmic vein
Annulus of Zinn
Lacrimal nerve (CN Va)
Frontal nerve (CN Va)
Optic canal
Trochlear nerve (CN IV)
Optic nerve (CN II)
Superior division of the oculomotor nerve (CN IIIs)
Ophthalmic artery
Nasociliary nerve (CN Va)
Inferior division of the oculomotor nerve (CN IIIi)
Abducens nerve (CN VI)
Ganglionic branches (from pterygopalantine ganglion to maxillary nerve))
Inferior ophthalmic veins
Infra-orbital nerve (CN Vb)
Zygomatic nerve (CN Vb)
Infra-orbital artery
Infra-orbital vein
Inferior orbital fissure

Fig: 13

Spaces of Orbit 00:12:32

- Intraconal space: a.k.a Retrobulbar Space
- The extraconal space: peripheral surgical space, contains the lacrimal gland, the superior oblique muscle and trochlea, and nerves and vessels in the extraconal orbital fat
- The subperiosteal space is a potential space between the orbital bones and periosteum lining.
- Tenon's capsule: which surrounds all but the anterior portion of the eye, is the bloodless space in which enucleation and scleral buckle procedures are performed.

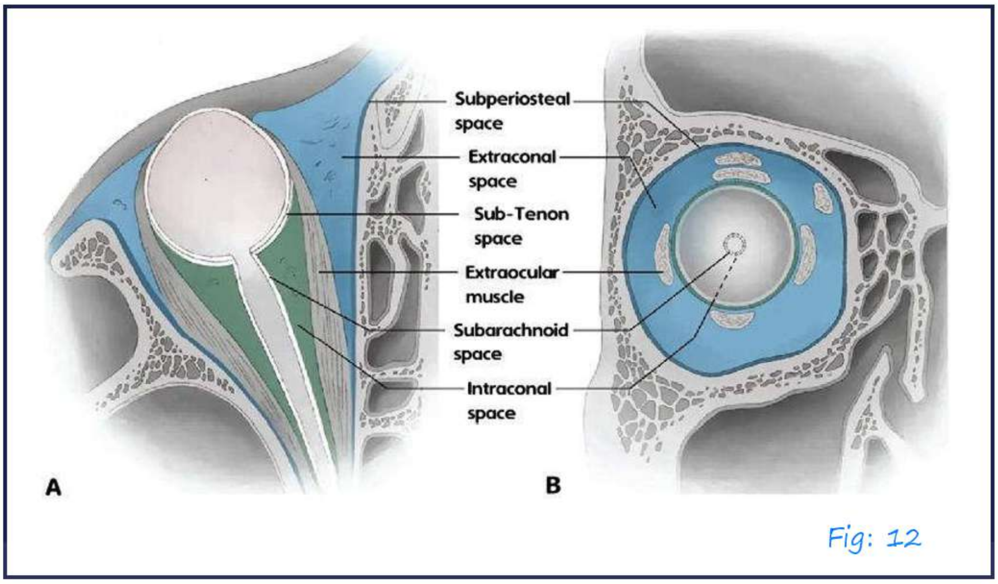

Subperiosteal space
Extraconal space
Sub-Tenon space
Extraocular muscle
Subarachnoid space
Intraconal space
A
B

Fig: 12

Supports of Orbit, eyeball 00:14:55

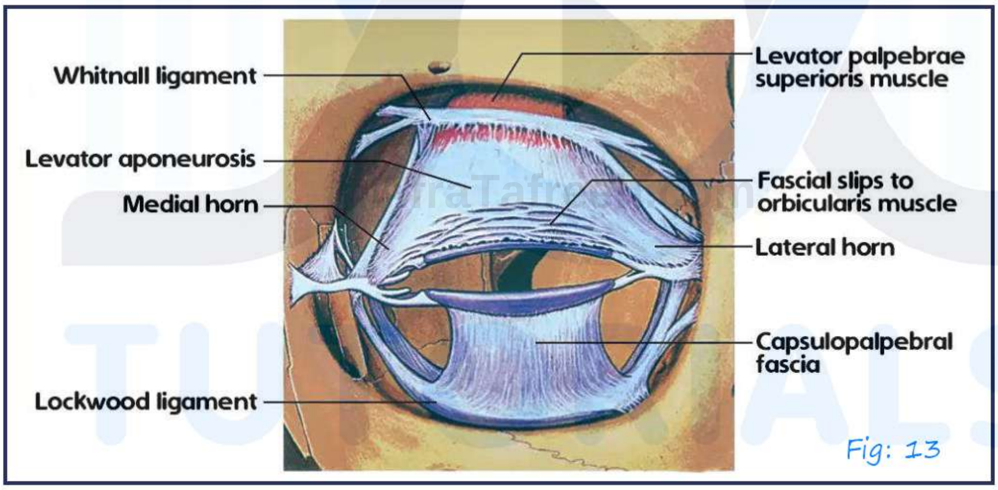

Whitnall ligament
Levator aponeurosis
Medial horn
Lockwood ligament
Levator palpebrae superioris muscle
Fascial slips to orbicularis muscle
Lateral horn
Capsulopalpebral fascia

Fig: 13

- Whitnall ligament
- Lock wood ligament
- The lower part of the bulbous sheath is known as the suspensory ligament of lockwood.
- Blends with the lateral canthus and the lateral check ligament and travels laterally to medially.

- Medially, blends with the medial canthus and the medial check ligament, attaching to the medial orbital wall at the lacrimal crest.
- The ligament encloses the inferior oblique and inferior rectus muscles, providing support and preventing downward displacement of the globe.

PROPTOSIS

Proptosis 00:00:15

- Forward protrusion of eyeball >21mm from lateral orbital rim

(or)

- Difference between 2 eyes > 2mm

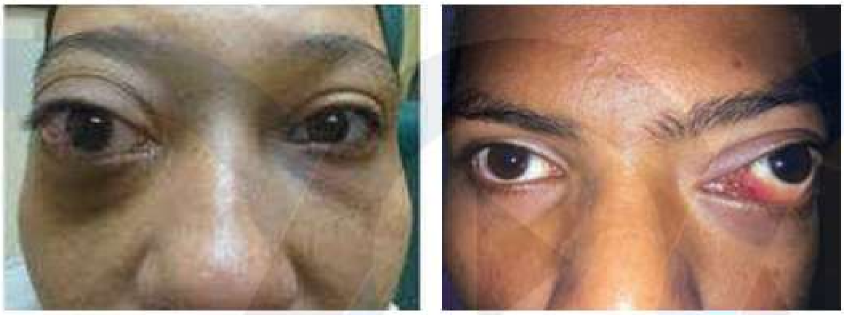


Fig: 1

- Dystopia -displacement of eye ball

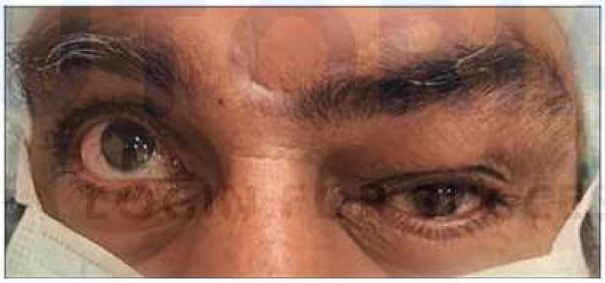


Fig: 2

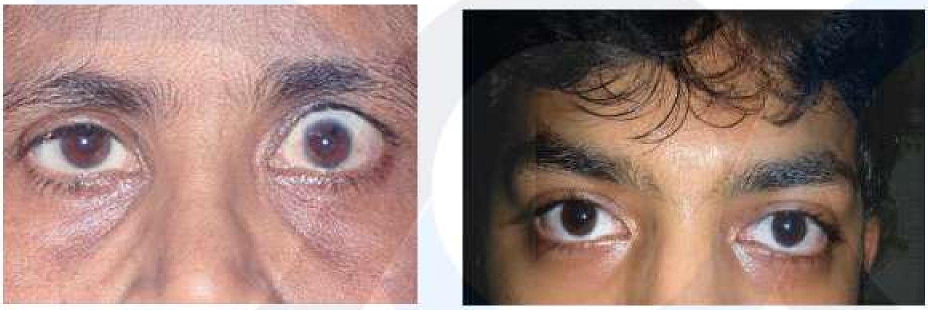
Dystopia- Frontal sinus mucocele

Pseudo Proptosis 00:04:50

- Some kind of facial abnormality that looks like proptosis
- In same eye causes
  - Axial myopia

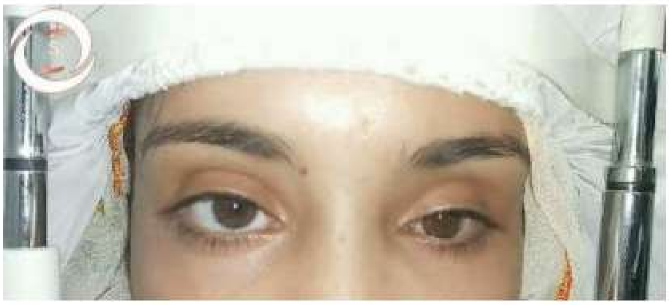
  - Buphthalmos
  - Retraction of lid
- Normal position of lid

Fig: 3



LE retraction RE high myopia Fig: 4

- Causes in contralateral eye
  - Enopthalamus /sunken eye
  - Blow out Fracture/Fracture of floor of orbit



Pseudo proptosis Fig: 5

DOT TUTORIALS LOGIN FOR SUCCESS

Measurement of Proptosis 00:08:10

Exopthlamometer

- Exophthalmos → Thyroid induced proptosis
  - Amount of proptosis

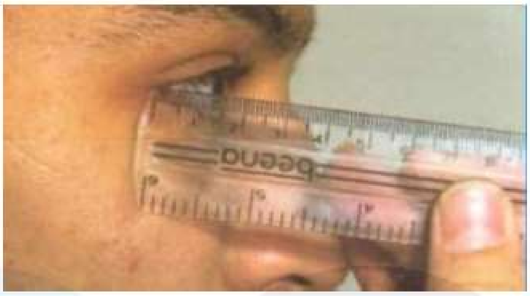


Fig: 6

Hertel's Exopthalamometer

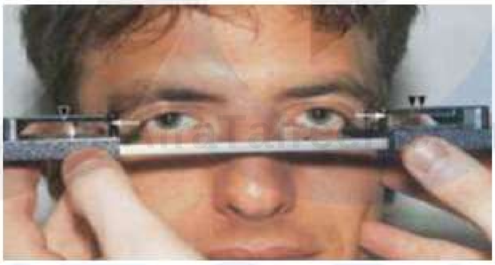


Fig: 7

Leudde's

- More accurate then Hertel's in presence of facial asymmetry
- Children

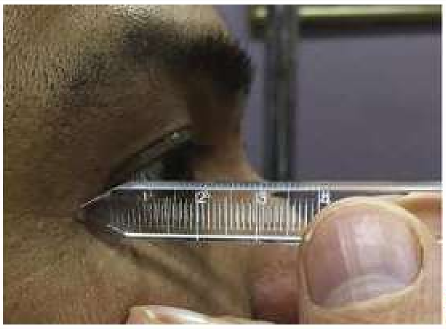


Fig: 8

Naugle's

- Measures the difference between two eyes rather than absolute measure
- It is prefered in the presence of an orbital Fracture or after orbitorfomy

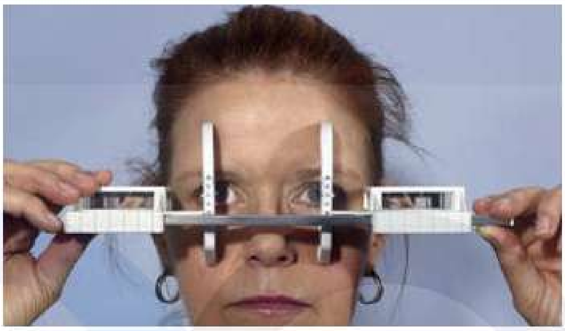


Fig: 9

Naffziger's Method:

- Relative protrusion can be observed by simply standing behind seated patient & gazing downward (tangentially) toward the chin from the fore head to assess protrusion of eye beyond the orbital rim

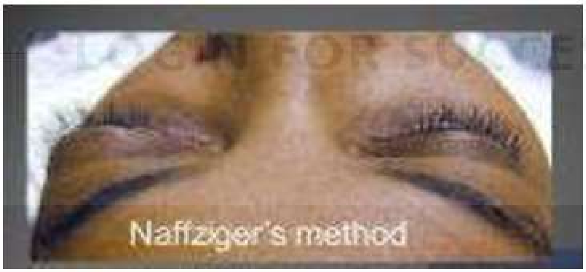


Fig: 10

Worms' Eye view;

- Similar to Naffziger's method
- Examiner examines up from below with the patients' head tilted back

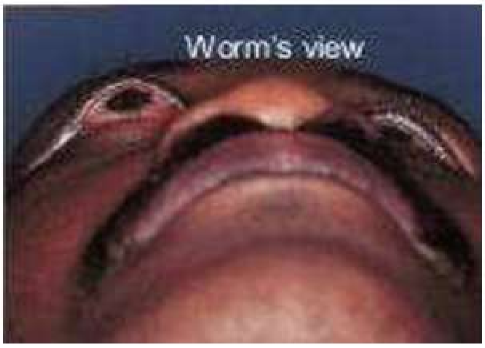


Fig: 11

Grading of Proptosis 00:13:10

- Mild – 22-24mm
- Moderate – 24-27 mm
- Severe - > 27 mm

History

- The Six P's described are
  - Pain
  - Progression
  - Proptosis type
  - Palpation
  - Pulsation
  - Periorbital changes

Causes of Proptosis 00:15:43

- Most common cause of U/L, B/L in adult
  - Thyroid Eye Disease

Thyroid Eye Disease

- Auto immune
- Increased secretion of GAGs

  ↓

  Imbibition of fluid

  ↓

  Increase Volume

  ↓

- Myofibroblasts
  - Soft tissues of orbit
  - Extra ocular muscles

Phases

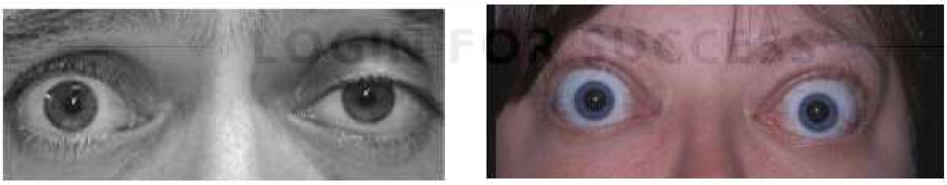
- Inflammation
- Fibrotic

  It can be hyper thyroid, hypothyroid, euthyroid state

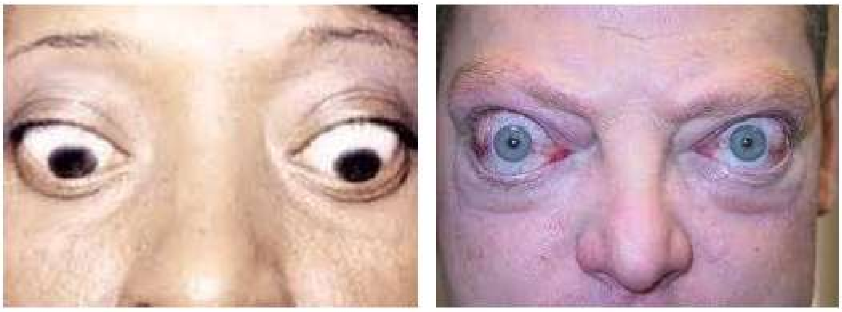
Clinical Features 00:18:58

Lid Signs/Conjunctiva

Signs	Features
Goldzeiher	Congestion of conjunctiva
Topolanski	Congestion of conjunctiva near recti insertion
Dalrymple	Lid retraction
Von graefe	Lid lags on looking down
Stellwag	Still look – less blinking
Jellinker	Pigmentation of eyelid
Enroth	Edema of lid
Rosenbach	Tremors on closed lids
Mobius	Decreased convergence
Kocher	Staring appearance



Dalrymple sign Fig: 12



Lid lag Von Graefe Fig: 13

EOM

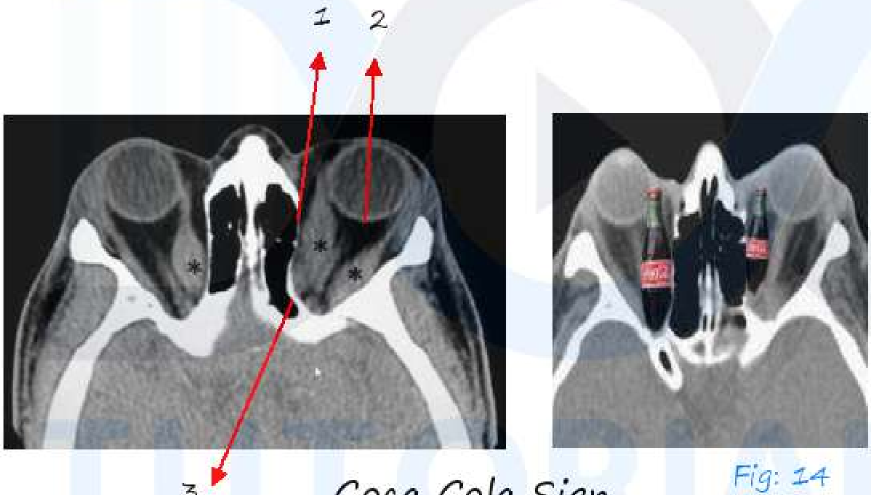
- 1st muscle to get affected is IR
- If IR is fibrotic → restrictive myopathy
  ↓
  Elevation is affected
- Sequence of muscle affected → Mnemonic IM SLO

I → IR

M → MR

SLO → SR LR (O → Oblique / Last)

- Tendons are always spared



Coca Cola Sign

Fig: 14

1. Muscle belly
2. Tendons are spared
3. Orbital apex

- Most common cause of lid retraction in adults
  - Thyroid
- NO SPECS classification: for thyroid eye disease

Class	Description
0	No symptoms and findings
1	No symptoms; there is only spasm in the upper eye-lid, eye opening increased
2	Swelling in the preorbital soft tissue
3	Proptosis (exophthalmos)
4	Involvement in extraocular eye muscles
5	Corneal involvement
6	Optic nerve involvement, visual loss in various levels

- The most current grading system of TED are the VISA
  - Vision
  - Inflammation
  - Strabismus
  - Appearance

Treatment 00:30:26

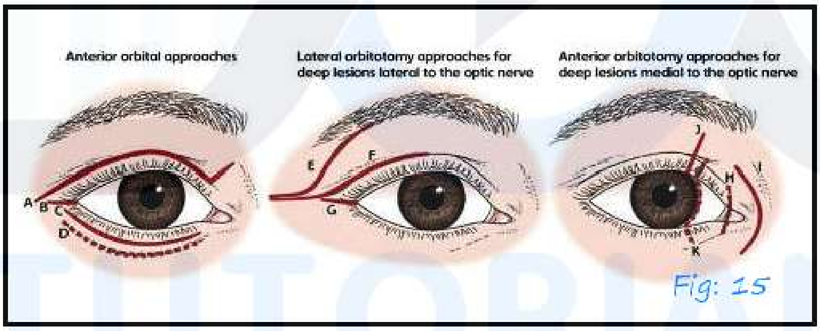
- **Tear supplements** – Prevent Excessive desiccation, corneal drying
- **Guanethidine** – For lid retraction

Systemic Steroids

- To decrease orbital inflammation oral prednisone in a dose of 1-1.5 -mg/kg can be given for a suggested maximum period of 2 months

Orbital Radiation

- Can be used alone or in conjunction with corticosteroids for decreasing inflammation
- Typical dose of 2000 cGy for each orbit 200 cGy/day given over period 10days
- Orbitotomy → to treat on compression


Anterior orbital approaches

Lateral orbitotomy approaches for deep lesions lateral to the optic nerve

Anterior orbitotomy approaches for deep lesions medial to the optic nerve

Fig: 15

- 2 wall decompression- Antero ethmoidal orbitotomy
- 3 wall decompression
- 4 wall decompression

Orbital Cellulitis 00:33:50

- Most common cause of Proptosis in children

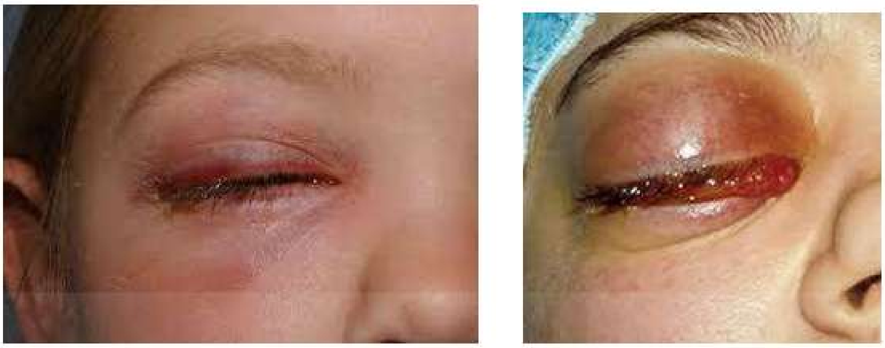


Fig: 16

- Pre septal cellulitis of eyelid which is separated by orbital septum
- Most common cause of orbital cellulitis is ethmoidal sinusitis
- Most of the time there is staph aureus infection

AmaYaheen.com

Chandlers staging 00:36:40

DOT TUTORIALS LOGIN FOR SUCCESS

Group 1	Pre-septal cellulitis	Inflammation and edema anterior to the orbital septum
Gruop2	Orbital cellulitis	Extension of inflammation to include the orbital contents posterior to the septum
Group 3	SPA Figure 3	Development of mucopurulent collection between the bony orbital walls and periorbital
Group 4	Orbital abscess	Development of mucopurulent collection/s within the orbital contents
Group 5	Cavernous sinus thrombosis	Development of retrograded phlebitis And coagulation of vascular contents extending up to the cavernous sinus giving rise to bilateral ophthalmic deficits

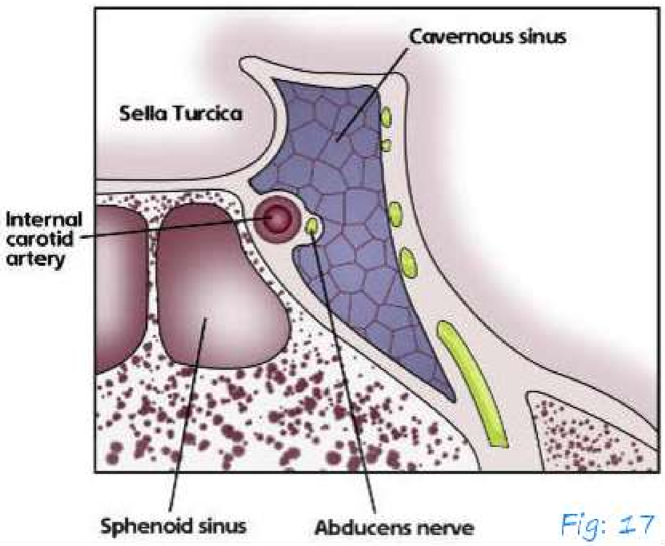
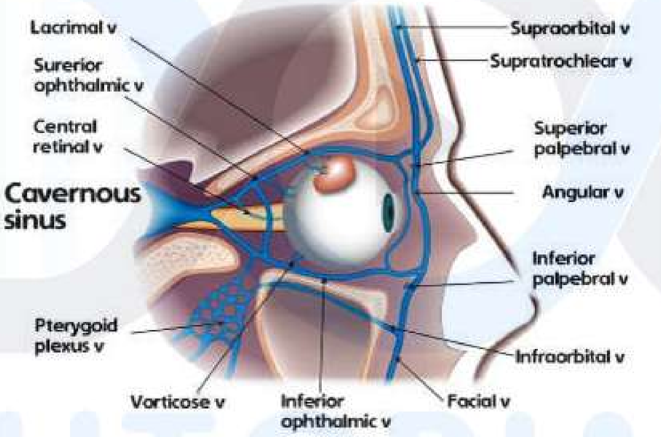
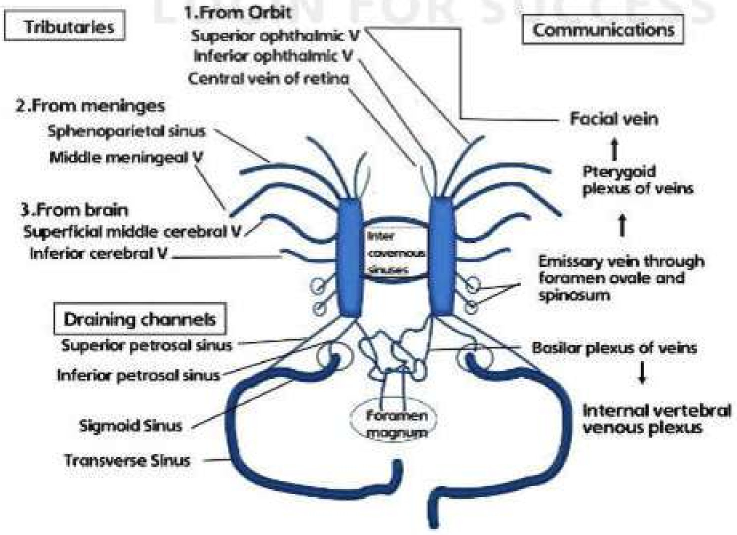

Cavernous sinus
Sella Turcica
Internal carotid artery
Sphenoid sinus
Abducens nerve

Fig: 17


Lacrimal v
Surerior ophthalmic v
Central retinal v
Cavernous sinus
Pterygoid plexus v
Vorticose v
Inferior ophthalmic v
Supraorbital v
Supratrochlear v
Superior palpebral v
Angular v
Inferior palpebral v
Infraorbital v
Facial v

cavernous sinus Fig: 18


Tributaries
1.From Orbit
Superior ophthalmic V
Inferior ophthalmic V
Central vein of retina
2.From meninges
Sphenoparietal sinus
Middle meningeal V
3.From brain
Superficial middle cerebral V
Inferior cerebral V
Draining channels
Superior petrosal sinus
Inferior petrosal sinus
Sigmoid Sinus
Transverse Sinus
Communications
Facial vein
Pterygoid plexus of veins
Emissary vein through foramen ovale and spinosum
Basilar plexus of veins
Internal vertebral venous plexus
Inter cavernous sinuses
Foramen magnum

cavernous sinus Fig: 19

Cavernous Sinus thrombosis

- 6th Nerve is 1st effected → so abduction is first affected
- Rapid proptosis
- Anterior segment signs

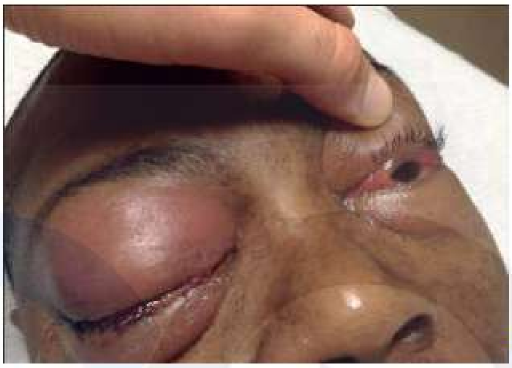


Fig: 20

- Loss of vision is a late feature
- It has inter cavernous sinus spread – B/L
- Fever, myalgia and other systemic signs
- Corneal sensations are decreased
- Edema in mastoid region, tenderness +

TUTORIALS LOGIN FOR SUCCESS AiraTaleem.com

Superior orbital fissure syndrome, Orbital apex syndrome 00:41:48

Superior orbital fissure Syndrome

- Infection is confined to SOF
- Vision is lost very late

Orbital Apex Syndrome

- SOF + Optic canal
- Vision loss earlier than proptosis

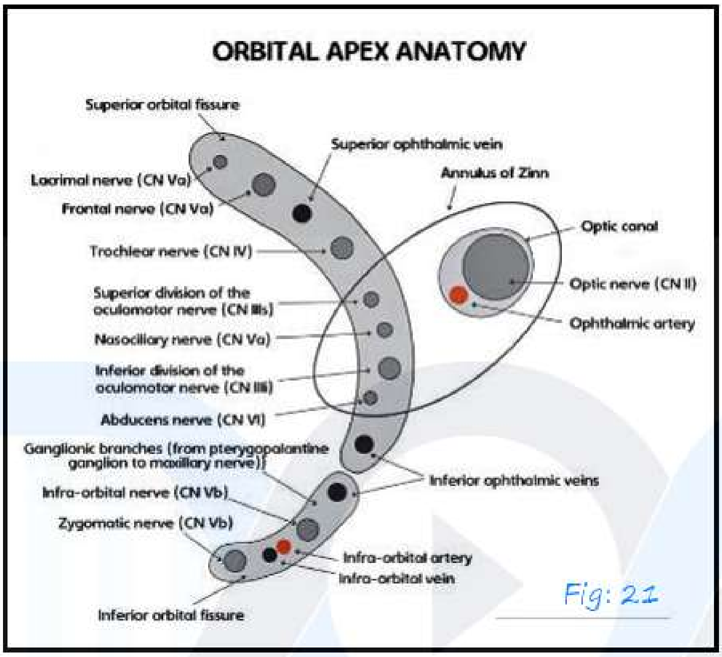

ORBITAL APEX ANATOMY
Superior orbital fissure
Lacrimal nerve (CN Va)
Frontal nerve (CN Va)
Trochlear nerve (CN IV)
Superior division of the oculomotor nerve (CN IIIs)
Nasociliary nerve (CN Va)
Inferior division of the oculomotor nerve (CN IIIi)
Abducens nerve (CN VI)
Ganglionic branches (from pterygopalatine ganglion to maxillary nerve)
Infra-orbital nerve (CN Vb)
Zygomatic nerve (CN Vb)
Inferior orbital fissure
Superior ophthalmic vein
Annulus of Zinn
Optic canal
Optic nerve (CN II)
Ophthalmic artery
Inferior ophthalmic veins
Infra-orbital artery
Infra-orbital vein

Fig: 2.1

Idiopathic Orbital Inflammatory disease (IOID)

00:43:11

- a.k.a Pseudotumor
- Non neoplastic, Non-Specific inflammation of orbital contents (dacryoadenitis, myositis)
- Usually seen in Young/Middle Ages presents with pain & proptosis
- Respond very well to steroids

Tolosa Hunt Syndrome 00:44:20

- Non-Specific granulomatous inflammation of orbital contents /Cavernous sinus, apex of orbit

Orbital Varices 00:45:13

- Inflammation of congenital malformed veins of orbits
- Orbital veins are devoid of valves
- When bending down/Valsalva maneuver pressure increased →proptosis increases
- This is intermittent proptosis
- It is seen as unilateral proptosis generally in children
- It is non-pulsatile/bruit
- Bag of worm kind of consistency of eyelid
- Sudden increased of proptosis may be there due to hemorrhage in varices
- Other caused of intermittent proptosis
  - Lymphangiomas
  - Hemangiomas

Carotico Cavernous Fistula 00:47:58

- Pulsative type of Proptosis
- Bruit +
- Pulsations can be felt in case of deficient roof of orbit
  - e.g.: NF

↓

Neurofibromatosis

Tumors Causing Proptosis 00:49:00

- Related to orbit/eye
- Most common orbital tumor in children → capillary hemangioma
- Most common orbital tumors in adults → cavernous hemangioma
- Most common malignant orbital tumor in child→ Neuroblastoma
- Most common Orbital tumors in adult → secondaries of Breast or lungs
- Most common primary malignant orbital tumors in child → Rhabdomyoma sarcoma
- Most common primary malignant orbital tumor in adults → non-Hodgkin lymphoma of orbit
- Most common Primary malignant intraocular tumor in child → Retinoblastoma

DESTRUCTIVE SURGERIES OF EYE

Evisceration 00:00:48

- Removal of contents of orbit
- Sclera
- Optic Nerve ⊢ Spared
- EOM

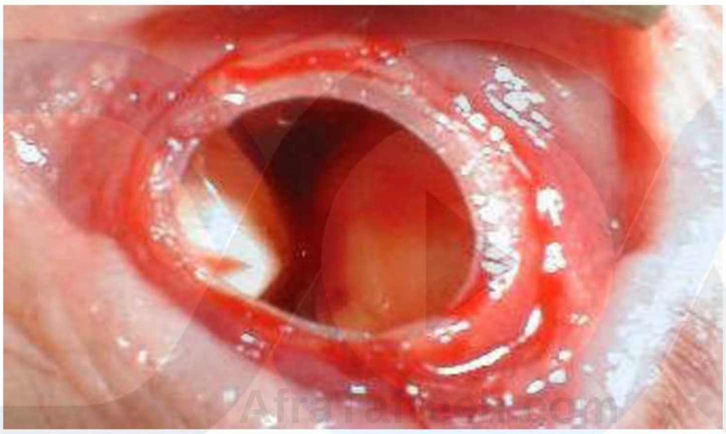


Fig: 1

- Indication → Pan ophthalmitis
  → Bleeding anterior staphyloma
- Contraindications → Tumors of eye ball
  → Sympathetic ophthalmia

Enucleation 00:03:00

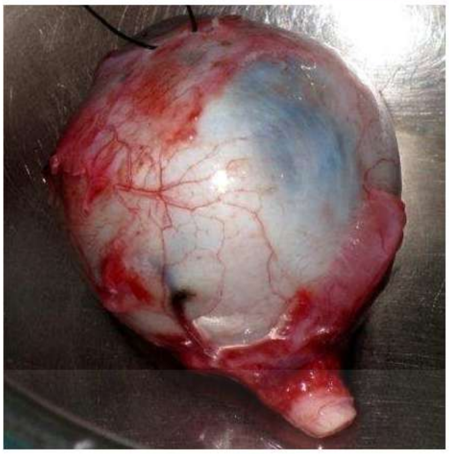


Fig: 2

- Total removal of globe with part of Optic Nerve
- Indications → Tumors of eyeball
  → Sympathetic ophthalmia

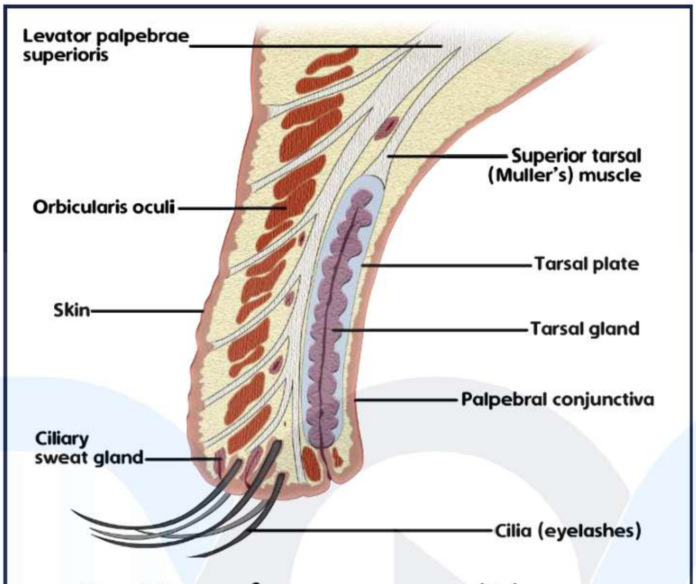
Exenteration 00:04:00

- Most Radical
- Removal of globe, Lid, Bones/walls
- Indication → Mucormycosis
  → Tumors of lid

TUTORIALS
LOGIC FOR SUCCESS

ANATOMY OF EYE LID

- Section of upper lid


Levator palpebrae superioris
Superior tarsal (Muller's) muscle
Orbicularis oculi
Tarsal plate
Skin
Tarsal gland
Palpebral conjunctiva
Ciliary sweat gland
Cilia (eyelashes)

Section of upper eye lid Fig: 1

Layers of eyelid 00:00:28

- Outermost – skin
- Inner most – Conjunctiva
- Skin – Thinnest skin of body
- Sub cutaneous tissue
  - Loose connective tissue
  - Rich in elastic fibers
  - No fat
  - Applied anatomy- fluid from oedema or hemorrhage rapidly engorges the loose subcutaneous eyelid tissue
- Muscle of protraction- Orbicularis oculi

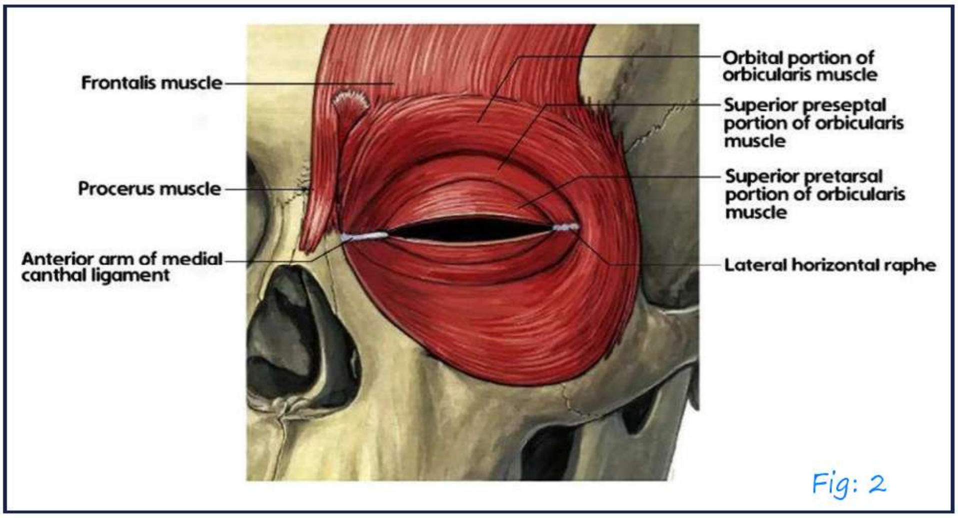

Frontalis muscle
Procerus muscle
Anterior arm of medial canthal ligament
Orbital portion of orbicularis muscle
Superior preseptal portion of orbicularis muscle
Superior pretarsal portion of orbicularis muscle
Lateral horizontal raphe

Fig: 2

- Orbicularis oculi
  - Striated muscle, concentrically arranged
  - Orbital, Palpebral
  - orbital part- Orbital process of frontal bone and Frontal process of maxillary bone
  - pretarsal part
  - Deep origin: Posterior lacrimal crest (Forms Horner's muscle)
  - Superficial origin: Anterior limb of medial canthal tendon

    preseptal part: Upper and lower border of medial canthal tendon to Lateral palpebral raphe

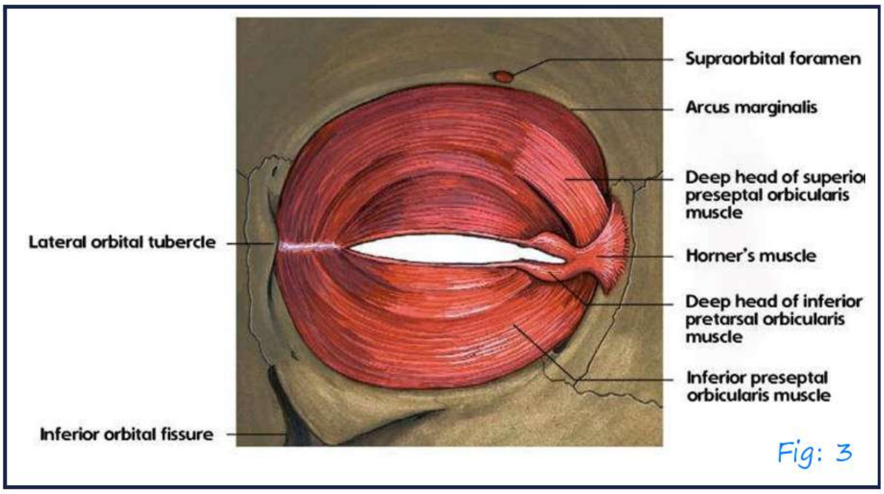

Supraorbital foramen
Arcus marginalis
Deep head of superio preseptal orbicularis muscle
Lateral orbital tubercle
Horner's muscle
Deep head of inferior pretarsal orbicularis muscle
Inferior preseptal orbicularis muscle
Inferior orbital fissure

Fig: 3

- Horner's Muscle – Pre-septal part of orbicularis which encloses lacrimal sac

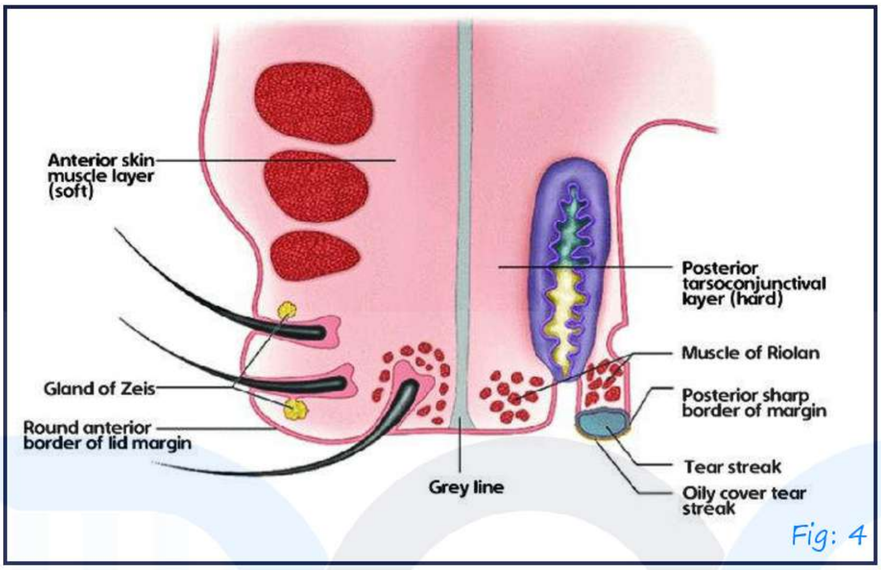

Anterior skin muscle layer (soft)
Gland of Zeis
Round anterior border of lid margin
Grey line
Posterior tarsoconjunctival layer (hard)
Muscle of Riolan
Posterior sharp border of margin
Tear streak
Oily cover tear streak

Fig: 4

- Muscle of Riolan– part of orbicularis going towards lid margin
- Orbicularis oculi basically help in closure of lid

- Orbital septum

TUTORIALS
LOGIC FOR SUCCESS

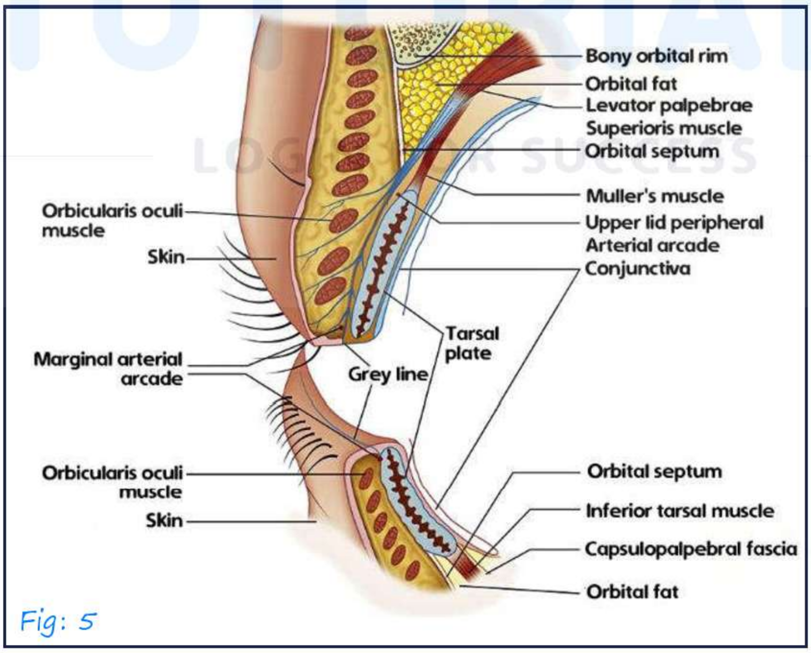

Bony orbital rim
Orbital fat
Levator palpebrae Superioris muscle
Orbital septum
Muller's muscle
Upper lid peripheral Arterial arcade
Conjunctiva
Orbicularis oculi muscle
Skin
Tarsal plate
Marginal arterial arcade
Grey line
Orbicularis oculi muscle
Skin
Orbital septum
Inferior tarsal muscle
Capsulopalpebral fascia
Orbital fat

Fig: 5

- Tarsal plate

Tarsal gland - Meibomian gland

- Orbital fat
- Muscle of retraction – LPS + Mullers muscle complex

↓

- LPS→ origin – Lesser wing of sphenoid
- Insertions
  - Anterior border of tarsal plate
  - Fornix
  - Crease of upper lid
  - Medial canthal tendon
  - Lateral canthal tendon
- Horizontal diameter is 40 mm
- Vertical diameter is 15 mm
- LPS - action → Chief elevation of upper lid
- Mullers muscle → Origin – Posterior fibers of LPS
  → Insertion – Upper border of tarsal plate

DOI TUTORIALS

- Grey line

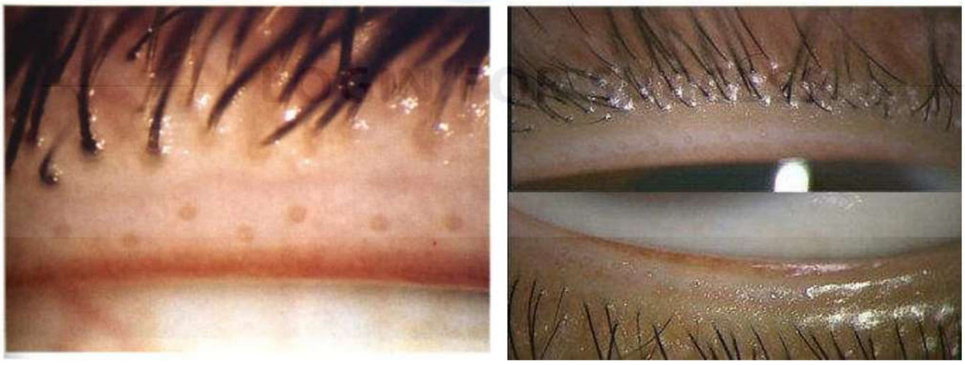


Fig: 6

- Divides eyelid into tow lamella
  - Anterior – Eyelashes
  - Posterior - Opening of meibomian gland

EYELID PATHOLOGIES

Ptosis 00:00:10

- Abnormal drooping of upper eye lid
- Congenital
  - Simple – most common (Due to abnormality in development of LPS)
  - Complicated
    - LPS + SR muscle problem
    - 3^{rd} C.N misdirection syndromes
- Acquired

  a. Neurogenic: 3^{rd} C.N Palsy, horners syndrome

  b. Myogenic: Muscular dystrophy, Myasthenia gravis

  c. Aponeurotic / senile ptosis: Dehiscence of LPS

  d. Mechanical – mass / tumor of upper lid.

Note:

- Muller's muscle also causes 1-2mm elevation
- Frontalis also has some contribution to elevation

Syndromes / Phenomenon

1.Marcus gun jaw winking phenomenon (HGJWP)

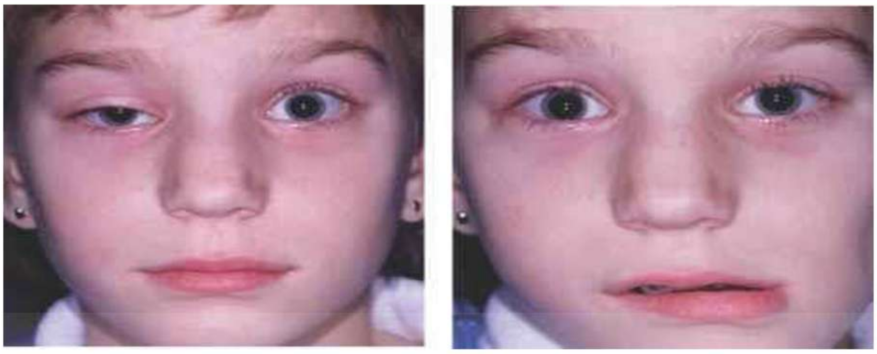


Fig: 1

- Misdirection of V C.N

  LPS is normally innervated by 3rd C.N, but in this condition it is innervated by 5th CN.

  I/L pterygoid is also innervated by 5th nerve

  Therefore, co-contraction of LPS + pterygoid occur

  ↓

  Causing elevation of eye on moving jaw to opposite side

2.Blepharo phimosis, ptosis epicanthus syndrome (BPES):

↓ ↓ ↓
Small palpebral severe extra lid fold skin near medial
Aperture congenital canthus (epicanthus inversus)

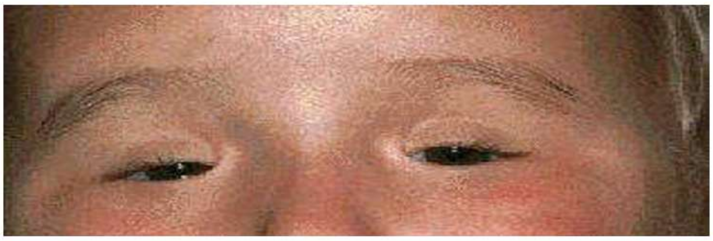


Fig: 2

It can be associated with telecanthus, eury blepharon, high thick arch with heavy eyebrow

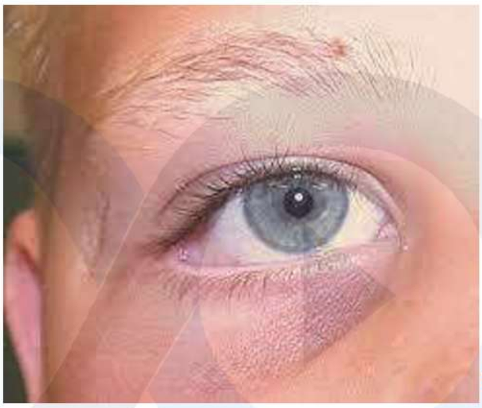


Fig: 3

Two types

Type 1: It is associated with premature ovarian failure

Type 2: No systemic association

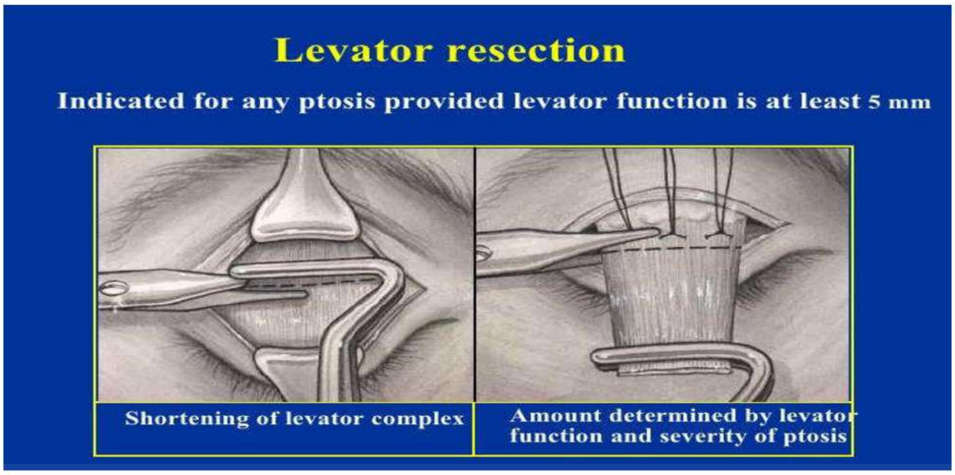
Management

- Crutch glasses

Surgery

1.LPS Resection – if muscle action is fairly good

EVERBUSCH	Blaskowics
Skin approach	Conjunctival approach.



Levator resection

Indicated for any ptosis provided levator function is at least 5 mm

Shortening of levator complex

Amount determined by levator function and severity of ptosis

Fig: 4

2.Sling surgery / Brow suspension / fascia lata (autologous):

Indication: Bilateral severe congenital ptosis and LPS action is poor

Prolene / silicone can be used as sling in place of fascia lata.

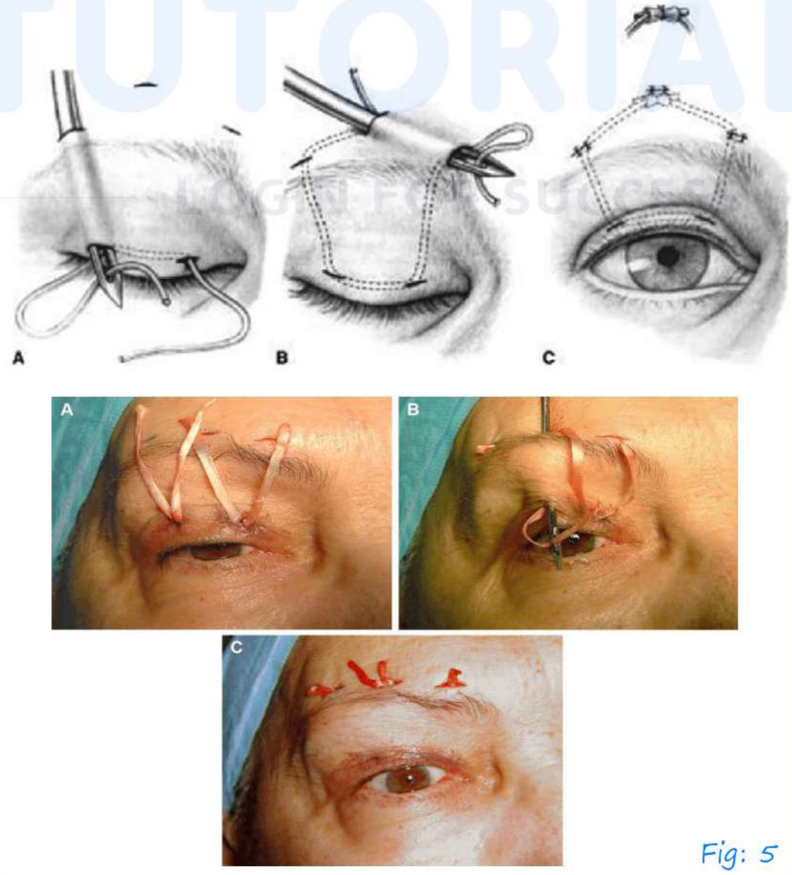


Fig: 5

3.Horner's syndrome ptosis: Fasanella servant surgery → Tarso mullerectomy,

Note

Epiblepharon: Extra horizontal lid skin, mostly on lower eyelid

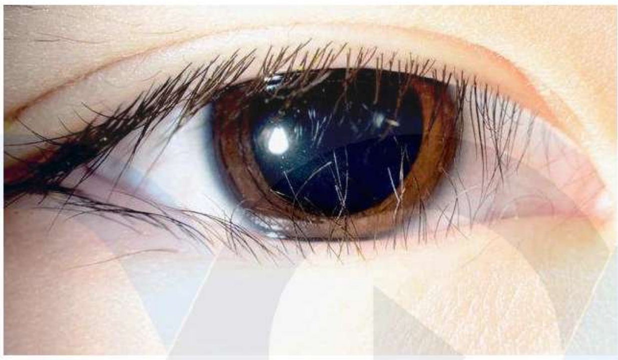


Fig: 6

Infections of eyelid 00:14:40

1.Meibomian gland

- Situated in tarsal plate
- Opening at posterior part of lid margin

2.Gland of zeis: situated at base of eye lash

3.Gland of moll: Situated in between two eye lashes

TUTORIALS LOGIN FOR SUCCESS

STYE / Hordeolum externum 00:16:02

- Acute painful suppurative inflammation of zeis and moll gland mostly caused by staph aureus
- Pus points towards eye lash

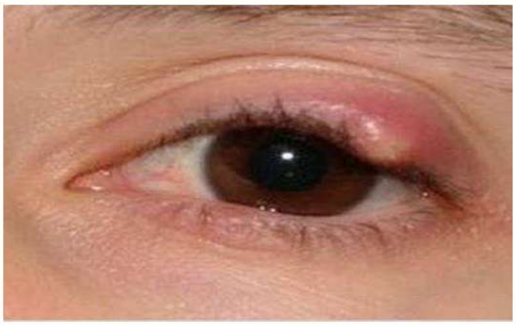


Fig: 7

Treatment

- Anti-inflammatory
- Antibiotics
- Hot packs
- Drain the pus – epilation

Chalazion 00:18:09

- Painless, chronic, non-suppurative granulomatous inflammation of meibomian gland
- Prominent point away from eye lash

TUTORIALS LOGIN FOR SUCCESS

Treatment

Incision and curettage →

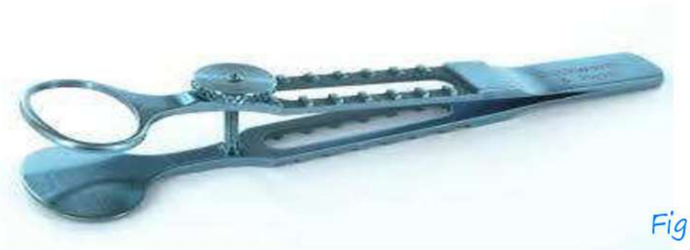


Fig: 8

- On conjunctival side
- Vertical incision to prevent damage to ducts

Internum hordeolum

- Acutely inflamed chalazion

  Treatment

  - Initially conservative with anti-inflammatory and antibiotics
  - Final treatment – incision and curettage

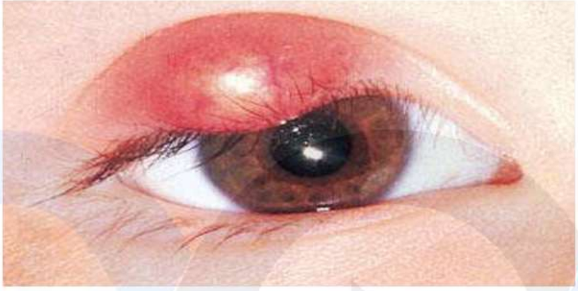


Fig: 9

Picture questions 00:20:47

- SymBlepharon: Adhesions between palpebral and bulbar conjunctiva seen in burns, chemical injuries, SJS, trachoma, ocular phemphigoid

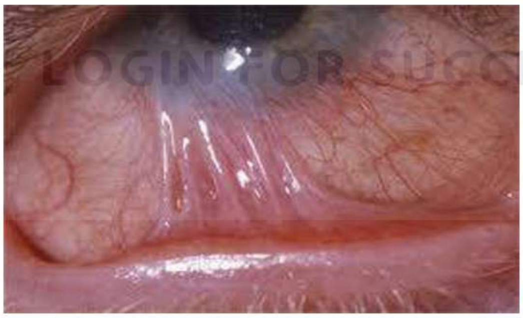


Fig: 10

- Ankylo Blepharon: Adhesion of upper and lower eyelid
  - Congenital condition

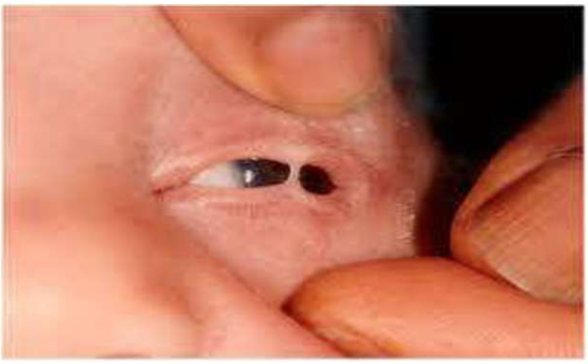


Fig: 11

- Entropion: Inward rolling of lid margins

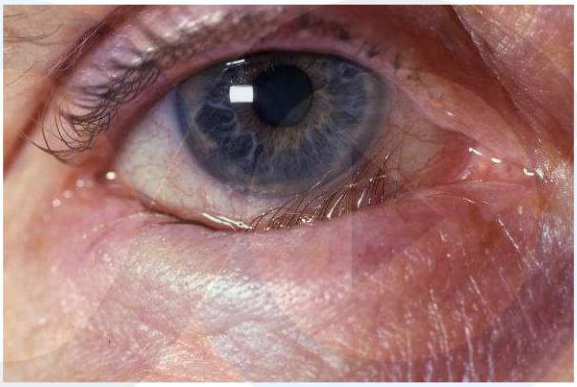


Fig: 12

4 types

C: Congenital

C: Cicatricial

S: Spastic – Due to irritation

S: senile – most common type affecting lower eye lid in old people

- Lid laxity
- Pretarsal is over rided by pre septal orbicularis

Management of entropion:

- Senile: Jones surgery, modified wheelers, Weiss surgery
- Cicatricial: Tarsal fracture- Burrows operation
- Spastic: botox toxin

Severe case
Burrow operation or tarsal fracture

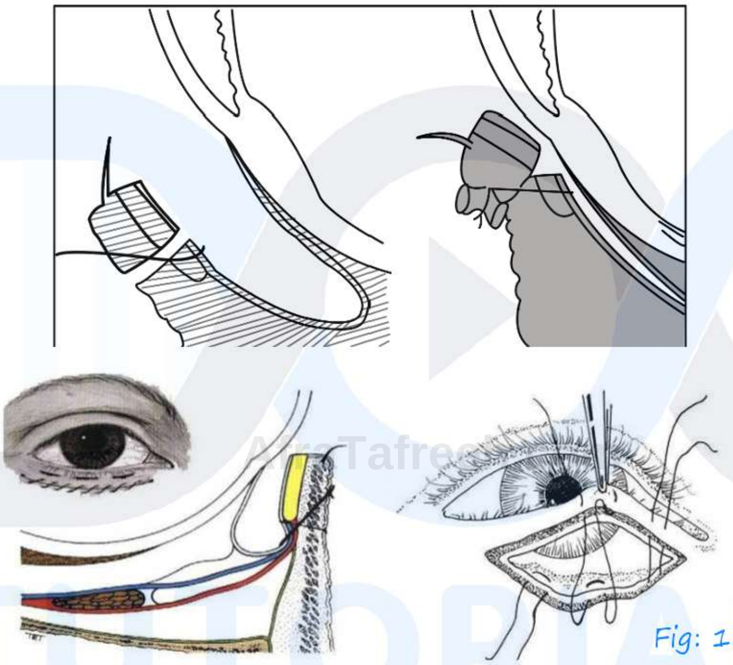


Fig: 13

DOC TUTORIALS
LOGIN FOR SUCCESS

Ectropion 00:25:07

Outward rolling of lid margin

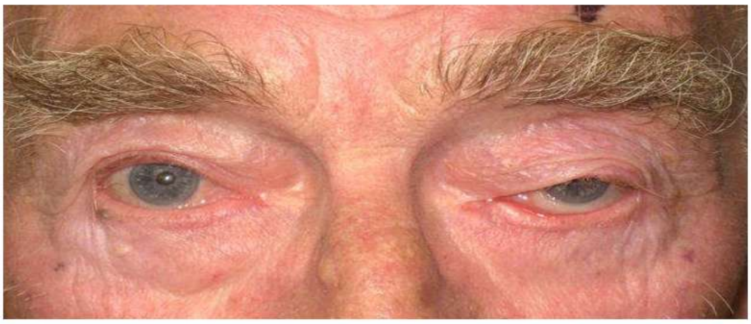


Fig: 14

1.c/o Epiphora

Types

- Paralytic
- Senile – most common type involving lower lid in old people
- Cicatricial

Treatment

- Medial conjunctivoplasty

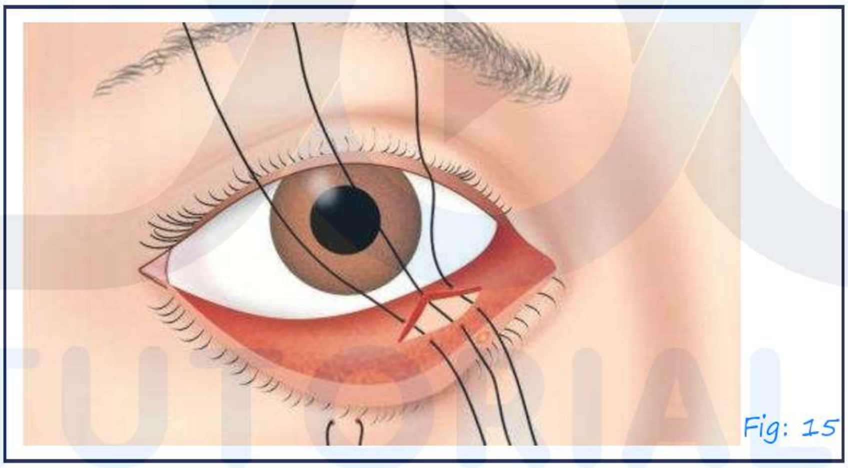


Fig: 15

TUTORIALS LOGIN FOR SUCCESS

- Horizontal lid shortening

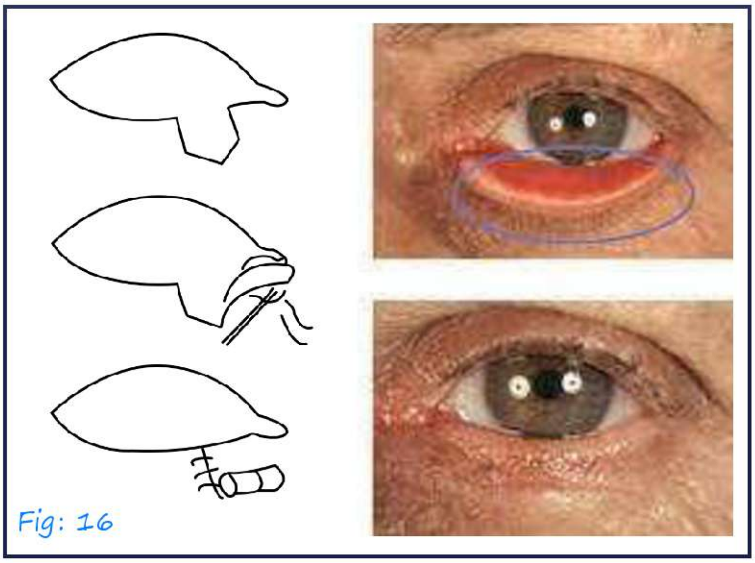


Fig: 16

- Bryon smith modified kuhnt szymanowski procedure
- V – Y plastsy
- Botox injection for paralytic ectropion induces temporary ptosis of upper eye lid

Trichiasis:

- Misdirected eye lashes

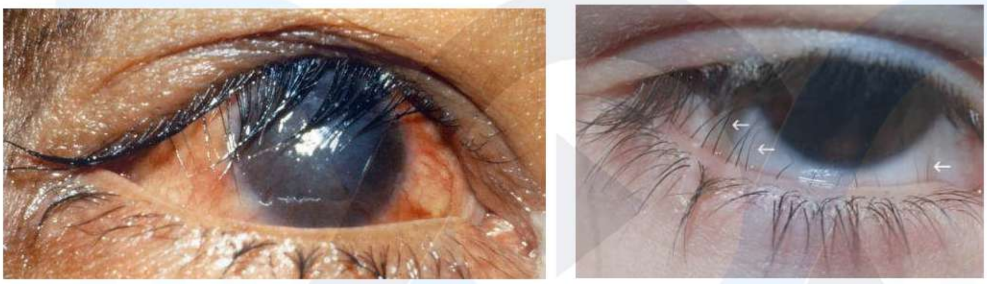


Fig: 17 Trichiasis Distichiasis

Distichiasis:

- Congenital anomaly with extra row of cilia
- Eye lashes grow towards cornea
- Autosomal dominant

Madarosis: Loss of eye lashes and eyebrows

- Blepharitis
- Infiltrating lid tumor
- Burns
- Radiotherapy
- Generalized alopecia
- Syphilis

- Leprosy
- Side effects
- Myxedema
- Trichotillomania

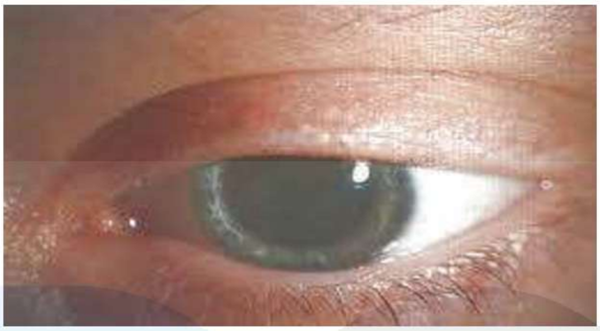


Fig: 18

Poliosis

- Premature greying of eye lashes

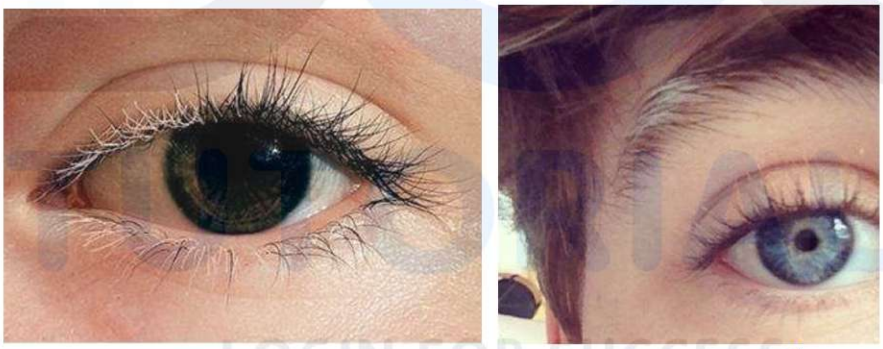


Fig: 19

Seen: VKH syndrome

Vitiligo

Marfan syndrome

Sympathetic opthalmia

Dermatochalasis

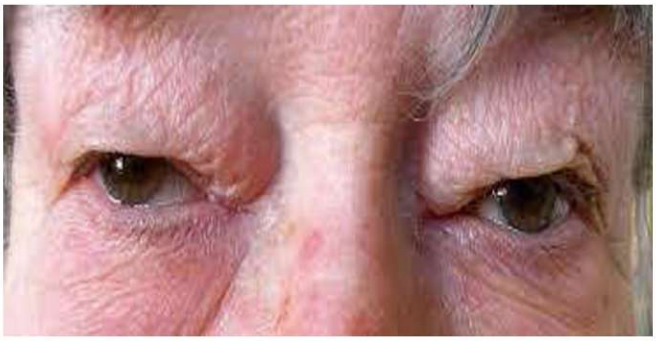


Fig: 20

- Protrusion / prolapse of ocular fat

Blepharochalasis

- Consists of recurrent bouts of upper eyelid edema associated with thinning, stretching and fine wrinkling of the involved skin
- This can result in eye lid skin redundancy

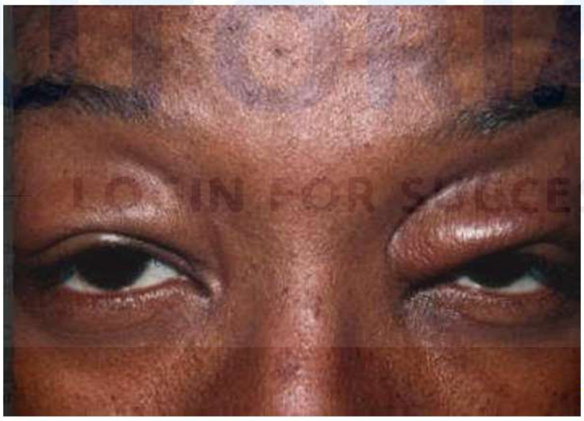


Fig: 21

- Seen in young people
- Rare condition

Blepharitis

Inflammation of lid margin

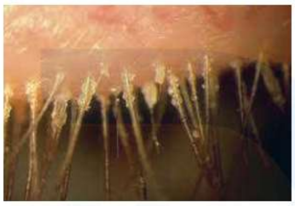
Anterior

posterior

Seiborrhoeic

↓

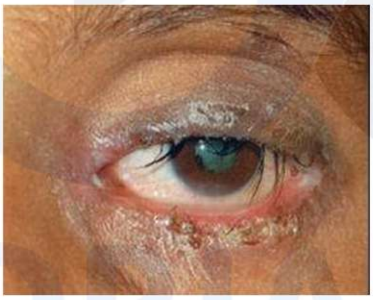
Soft scales over eye lashes



Staphylococcal / ulcerative

↓

- Associated with notching and scarring of lids
- Scales are hard and difficult to be removed



Posterior blepharitis

- Meibomian gland disease
- Pouting / secretions coming out from lid margin

Association: Roseacea

Treatment

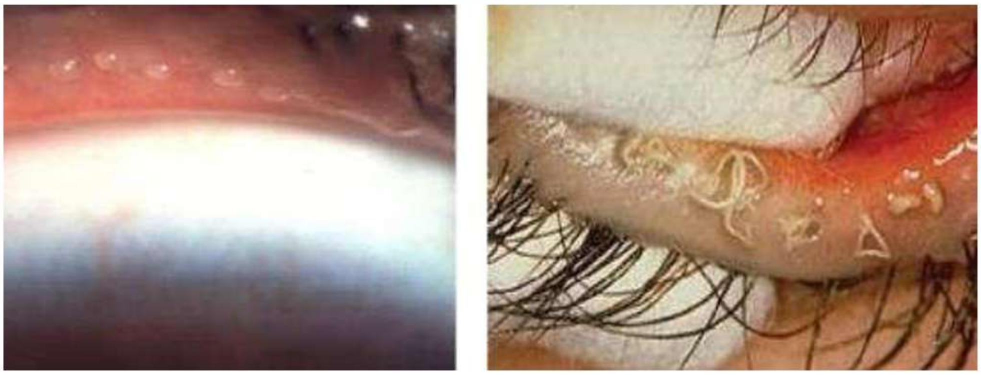


Fig: 22

- Lid scrubbing using baby shampoo
- Hot compression – to release the secretions
- Oral tetracyclines

LID Cancers 00:33:45

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1.Basal cell carcinoma

- Most common cancer
- Behave as rodent ulcer
- Most common location lower lid medial canthus

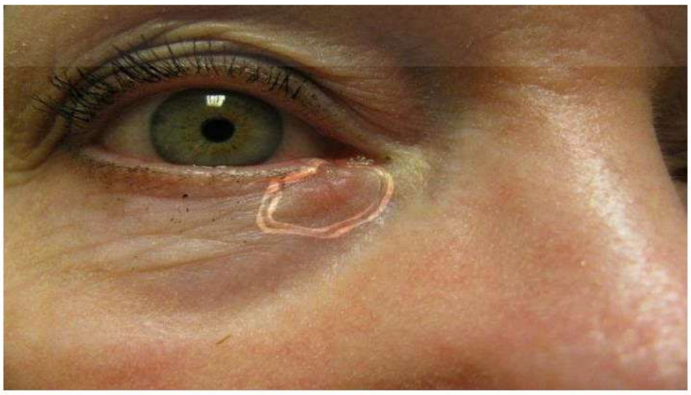

Fig: 23

2.Squamous cell ca

3.Sebaceous cell ca

H/O recurrent chalazion in elderly ⊕ in same place mainly in upper lid.

LACRIMAL APPARATUS & RESTRICTIVE SQUINT

Location: Superotemporal part of orbit → main lacrimal gland

Orbital
Separated by LPS
Palpebral part (more important)

- Responsible for reflex tear secretion
- Accessory lacrimal glands: Glands of Krauss and wolf ring
- Responsible for basal tear secretion

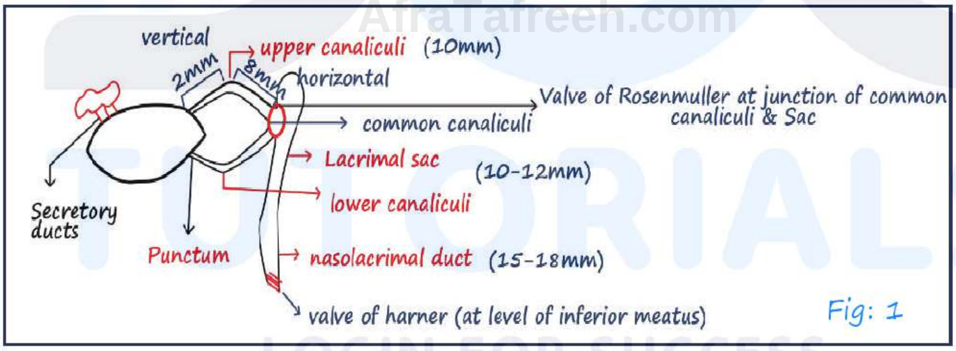
vertical
2mm
8mm
upper canaliculi (10mm)
horizontal
Valve of Rosenmuller at junction of common canaliculi & Sac
common canaliculi
Lacrimal sac
(10-12mm)
lower canaliculi
Secretory ducts
Punctum
nasolacrimal duct (15-18mm)
valve of harner (at level of inferior meatus)

Fig: 1

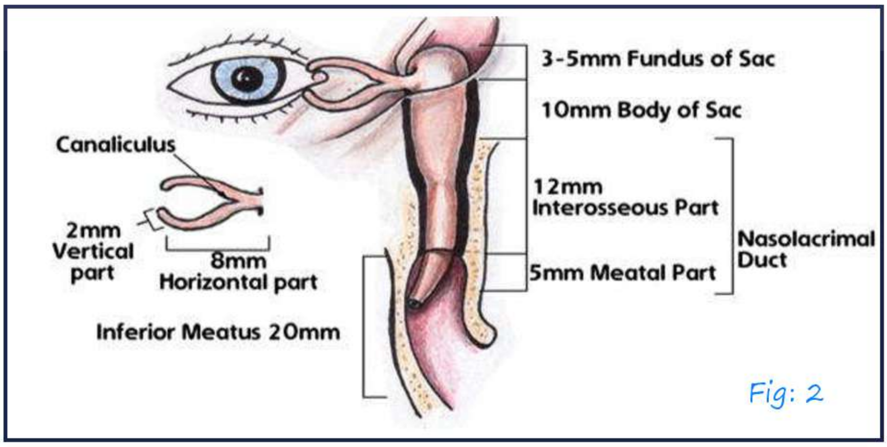
3-5mm Fundus of Sac
10mm Body of Sac
Canaliculus
12mm Interosseous Part
Nasolacrimal Duct
2mm Vertical part
8mm Horizontal part
5mm Meatal Part
Inferior Meatus 20mm

Fig: 2

- Lower punctum / lower canaliculi are very important: 80% of drainage of tear occurs here

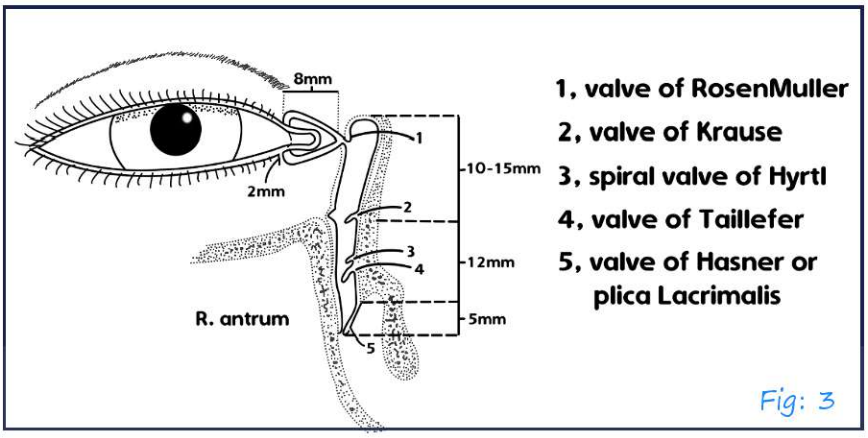
8mm
2mm
10-15mm
12mm
5mm
R. antrum
1
2
3
4
5
1, valve of RosenMuller
2, valve of Krause
3, spiral valve of Hyrtl
4, valve of Taillefer
5, valve of Hasner or plica Lacrimalis

Fig: 3

Lacrimal pump 00:04:52

- In relaxed state, the puncta lie in tear lake
- With eyelid closure, orbicularis muscle contracts
- Pretarsal orbicularis squeezes and closes puncta and canaliculi
- Preseptal orbicularis (Horners muscle) inserts into the lacrimal sac, pulls the lacrimal sac open, creating a negative pressure that draws the tears into the sac
- With eye lid opening, the orbicularis relaxes, puncta open, lacrimal sac collapses, propelling tears down the duct

Watering of eye 00:06:30

1. Hyper secretion → increase secretion of tears
2. Epiphora → watering of eye due to obstruction in drainage pathway

Eg: stenosis of punctum

Infections

Tumors of sac

polyp

Dacrocystitis 00:08:20

Inflammation of lacrimal sac

1.Acute: Acute painful suppurative inflammation of lacrimal sac

Caused by staph, streptococcus, pneumococcus

Treatment: - Hot packs

Anti-inflammatory

Antibiotics

Incision and drainage of pus

2.Chronic dacryocystitis: Painless inflammation of sac

Most common in females since they have narrow tract thus increase risk of obstruction and infection

C/O: Watering in eye.

1.Test: Regurgitation test

Press on lacrimal sac area – Regurgitation of fluid seen – than it is positive

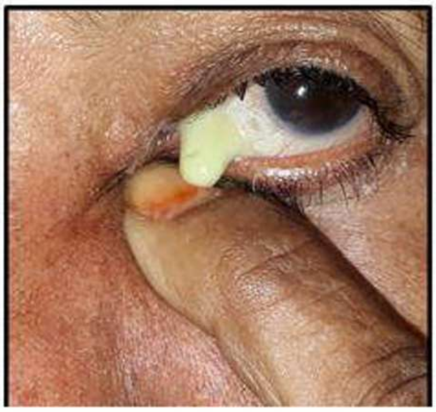

Fig: 4

2.Syringing: Done to know the level of block

After giving local anesthetic drops – punctum is dilated

↓

Syringing is done

Interpretation:

i. Done from lower punctum – No fluid comes out i.e. canal is patent

ii. Done from lower punctum – Regurgitation of fluid from opposite punctum → obstruction at common canaliculi

iii. Done from lower punctum → Regurgitation from same punctum

↓

Block at same canaliculus

iv. Done from lower punctum → Secretion of mucoid flakes from same) or opposite punctum after some time

↓

Block at nasolacrimal duct / lower part of sac

3.Probing

Done using bowman's probe

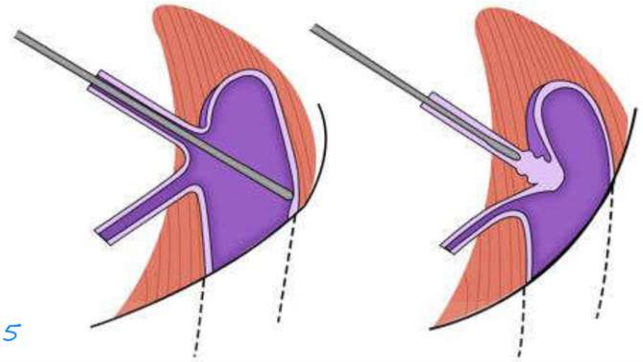

Fig: 5

Hard stop: Bone (i.e. Beyond lacrimal sac)

Soft stop: Canaliculi

4.Dye Disappearance test:

- A fluorescein strip is placed in conjunctival fornix, this leads to change in colour of tears
- Normally dye disappear in 5 min due to complete drainage
- Delayed disappearance of dye is significant for Epiphora/ obstruction

AfraTafreen.com
DOC TUTORIALS LOGIN FOR SUCCESS

5.Jones dye test

It is done in cases where epiphora is present and on syringing NLD is patent.

Two steps

- Step 1: Stain tear film with fluorescein dye. After anaesthetizing nasal mucosa anesthetic soaked cotton wool is placed in nose
  a. If cotton wool stained – The passage is patent, i.e., Jones dye 1 test is positive. Therefore, the cause of epiphora may be hypersecretion
  b. If no stain to cotton: - Jones dye 1 test is negative

  ↓↓

  Jones 2 test is done.
- Step 2: Syringing is done
  a. If cotton is stained – The passage has been made patent (Partial obstruction was present beyond sac i.e. Jones dye 2 test is positive
  b. If cotton is not stained – I.e., obstruction is before NLD and lacrimal sac jones dye 2 test is negative

  Pump failure / obstruction higher up in the passage
- Dacryocystography- Radiographic visualization of the lacrimal sac and associated structures after injection of a contrast medium – lipidol
- Example: Done in cases of trauma (or) when syringing and probing are not possible

Fig: 6

- Dacryoscintigraphy: It involves use of technetium 99m colloid / in some cases pertechnetate

  Procedure: Patient place their head in a support and chin rest and a single drop of 0.01ml is delivered to each eye. Imaging with a gamma camera done for 15 mins

  Treatment:

  In chronic dacryocystitis – DCR (Dacryocystorhinostomy done)

  ↓

  Creation of Passage between lacrimal sac and middle meatus of nose

- Congenital dacryocystitis
  - Obstruction at lower end of NLD
  - Most commonly membranous blocks
  - In majority (90%) of children opens on it's own due to canalization

    Management:

    <1 year: Criggler's massage (Done till child becomes 1 year old)

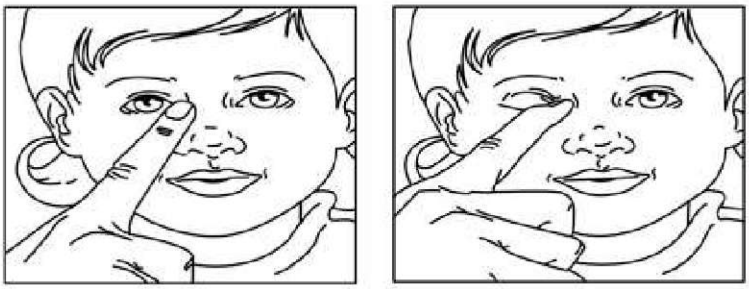

Fig: 7

>1 year: Syringing and probing

>4 years – DCR

Note

Dacryoadenitis: Inflammation of lacrimal gland

"S shaped upper eye lid seen"

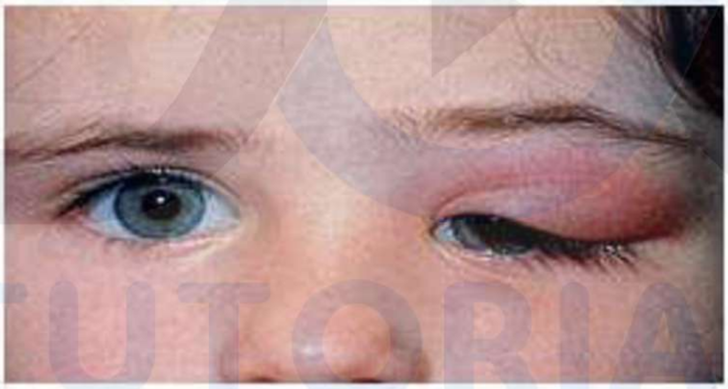

Fig: 8

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Healthy tears 00:31:04

- A complex mixture of proteins, mucin and electrolytes
- Antimicrobial proteins like lysozyme, lactoferrin is present
- Growth factors and suppressors of inflammation seen
- Soluble mucin secreted by goblet cells for viscosity
- Electrolytes for proper osmolarity

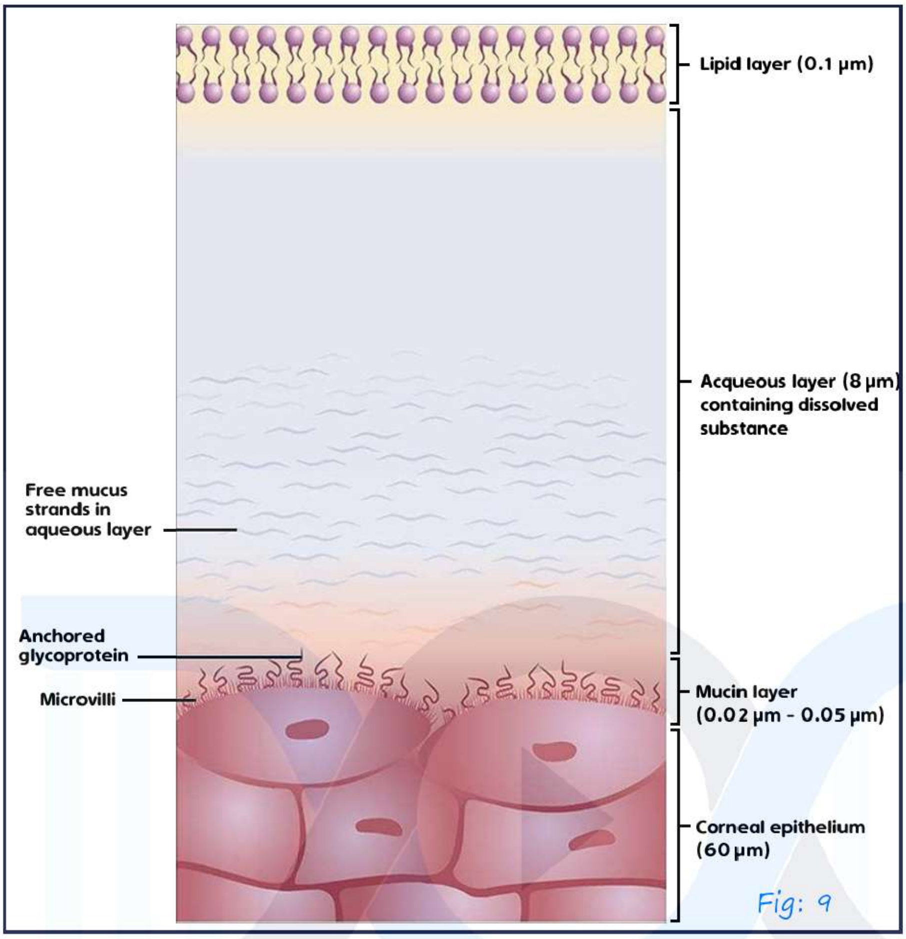

Lipid layer (0.1 µm)
Acqueous layer (8 µm) containing dissolved substance
Free mucus strands in aqueous layer
Anchored glycoprotein
Microvilli
Mucin layer (0.02 µm - 0.05 µm)
Corneal epithelium (60 µm)

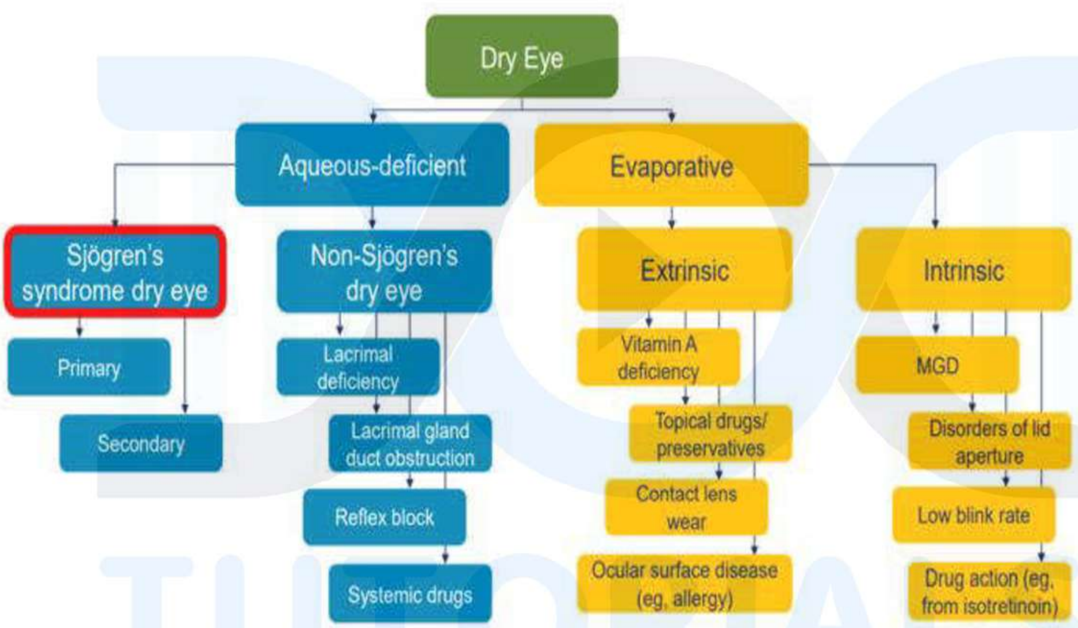
Fig: 9

Dry eye 00:33:40

Holly and kemp classification (1997)

Aqueous deficiency	Mucin deficiency	Lipid deficiency
KCS	Vitamin a deficiency	MGD
Sjogren syndrome	SJ syndrome	Posterior blepharitis
Age related hyposecretion	Ocular pemphigoid	Rosacea
Riley day syndrome	Burns	Bell's palsy
Congenital alacrimia	Chemical injuries	Contact lens wear
Lacrimal gland disorders	Trachoma	Atopic keratoconjunctivitis

- DEWS Definition (2007): It is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance and tear film instability with potential damage to the ocular surface
- It is accompanied by increase osmolarity of the tear film and inflammation of the ocular surface.


Dry Eye
Aqueous-deficient
Evaporative
Sjögren's syndrome dry eye
Non-Sjögren's dry eye
Extrinsic
Intrinsic
Primary
Secondary
Lacrimal deficiency
Lacrimal gland duct obstruction
Reflex block
Systemic drugs
Vitamin A deficiency
Topical drugs/ preservatives
Contact lens wear
Ocular surface disease (eg, allergy)
MGD
Disorders of lid aperture
Low blink rate
Drug action (eg, from isotretinoin)

Symptoms of dry eye:

- Grittiness / foreign body sensation
- Dryness

1.Test for dry eye

Slit lamp examination:

- Increase debris / mucin strands in tear film
- Inspection of tear meniscus at lid margin (TMH)
- Normal thickness – 0.5 to 1mm, convex

<0.5 mm – Tear deficiency

Severe cases – marginal tear meniscus is concave, small and absent

2.Schirmers test

Whatman filter paper 41 with (35 mm x 5 mm) is used

- Schirmers 1 test: Measure total (reflex + basal) tear secretion
  - Normal wetting: - 10-15mm
  - Dry eye
  - Mild: 9-14mm
  - Moderate: 4-8mm
  - Severe: <4mm
- Basal secretion test: To measure basal secretion by eliminating reflex tearing "using anesthetic drops" <5mm hyposecretion. (After 5 min)

Schirmer test 2

- To ascertain reflex secretion
- Measured after 2 min, after strips are installed, unanesthetized nasal mucosa is irritated. <15mm: failure of reflex secretion.
- Schirmer 1: BST (With anesthetic)
- Schirmer 2: Without anesthesia but with nasal stimulation – measures maximum basal and reflex secretion

Phenol red thread test:

Yellow colour thread impregnated with phenol red dye

↓

When tear comes in contact with thread

↓

Colour changes from yellow to red

> 10mm: normal tear production

T BUT (Tear film breakup time)

- Time taken for breakage of tear film is noted

  Normal: 25-30s

  <10s – tear film instability
- TBUT women < men
- It decreases in elderly

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Tear clearance rate

- Tear clearance rate (TCR)
- 5 µl of fluorescein instilled
- Residual dye measured in a Schirmer's strip at intervals of 1, 10, 20 and 30 minutes
- Presence of fluorescein in each strip is examined under blue light and compared to a standard scale / fluorometry
- Normal eyes – 0 after 20 minutes
- Dry eyes – delayed clearance (decrease turnover of tears).

Tear function index

- TFI = Schirmer test / TCR
- TFI <16 --- Possible Sjogren's Syndrome
- 16 < TFI < 96 --- Consistent with early tear deficiency
- TFI > 96 --- Normal
- Impression cytology – Determine the goblet cell density of bulbar and palpebral conjunctiva
- Tear film osmolarity: Normal: 295 to 309 m osm/litre elevated in dry eyes

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Treatment

- Tear substitutes
  1. Cellulose derivatives: Hydroxy propyl methyl cellulose, carboxy methyl cellulose
  2. Polyvinyl alcohol – increase persistence of tear, better in mucin deficiency
  3. Sodium hyaluronate – promotes conjunctival corneal epithelial healing
- Preservation of existing tears
  - Bandage contact lenses

    Reduction of tear drainage
  - Punctal occlusion

    a) Temporary: Collagen plugs are used which dissolve in 1-2 weeks' time

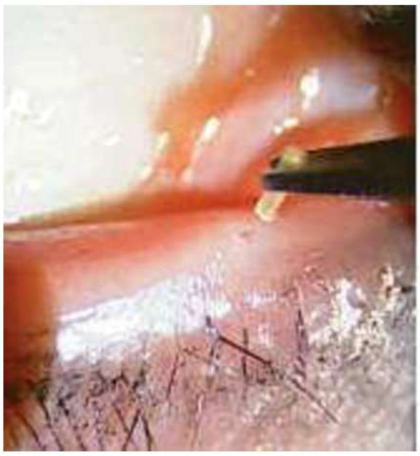


Fig: 10

b)Permanent: - done in Severe keratoconjunctivitis sicca

-Repeater Schirmer <2mm

- Treatment of diseases predisposing to dry eye

Other agents

- Autpologous serum drops
- Corticosteroids → decrease inflammation
- cyclosporin's
- Mucolytics – acetylcysteine
- Oral pilocarpine -increase secretion
- Tarsorrhaphy.

OPTICS OF NORMAL EYE, REDUCED EYE & VISUAL ACUITY ASSESSMENT

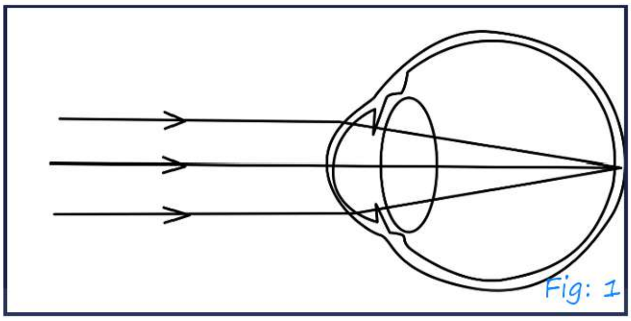


Fig: 1

- Emmetropia
- When parallel rays of light without any accommodation come to fovea on eye
- Surface with maximum refractive capacity → cornea

Refractive indices

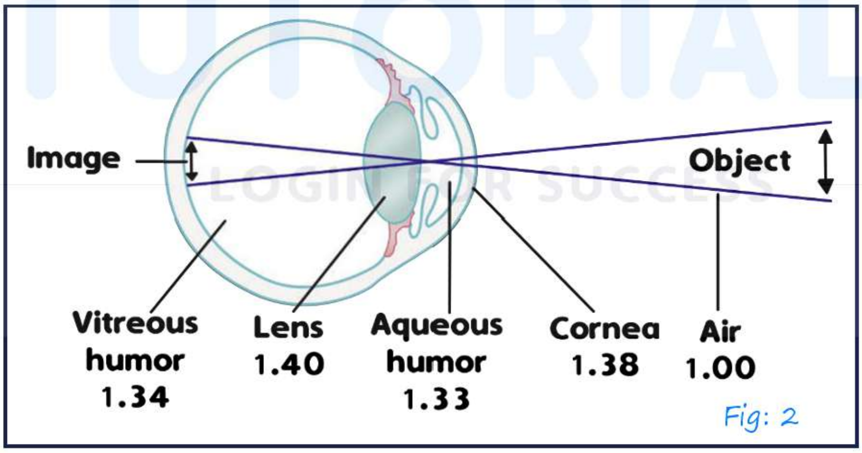


Image
Object
Vitreous humor 1.34
Lens 1.40
Aqueous humor 1.33
Cornea 1.38
Air 1.00

Fig: 2

Power → + 60D eye

+ 43D cornea   +17D Lens

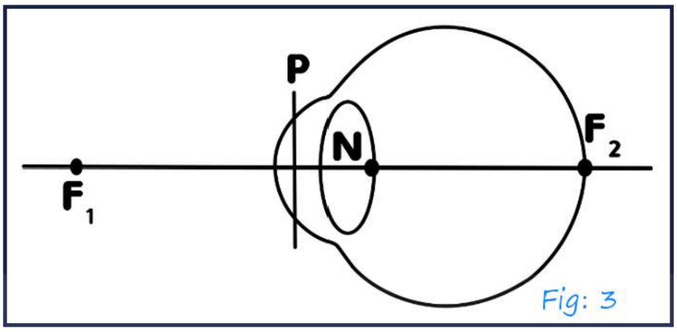


Fig: 3

Principal focal point	1.35 mm
Nodal point	7.08mm
First focal point	-15.7mm
Second focal point	24.13mm

- Listings reduced eye
- Anterior surface of cornea as the total refractive body → Most X
- Nodal point → 1.08mm from anterior surface of cornea – Near posterior part of lens

↓

Part where rays of light goes undeviated

↓

reason for PSC to gaze glare

- First focal point → Infront of cornea
- 2nd focal point → Behind cornea

Visual acuity assessment 00:08:20

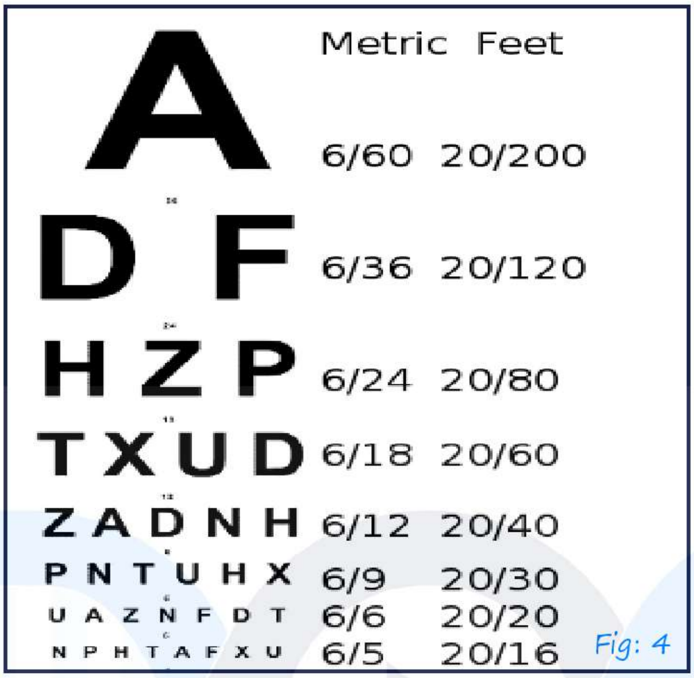

Metric Feet
A 6/60 20/200
D F 6/36 20/120
H Z P 6/24 20/80
T X U D 6/18 20/60
Z A D N H 6/12 20/40
P N T U H X 6/9 20/30
U A Z N F D T 6/6 20/20
N P H T A F X U 6/5 20/16

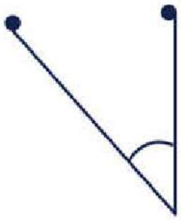
Fig: 4

- Sellen's chart
- Distance at 6meter (Western – 20feet)
- 6/60 → Numerator → Distance of patient → 6

  → Denominator – Distance of Normal individual from chart
- Close 1 eye and examine
- 6/24 → Any individual could have seen this line from 24m

Visual activity

- Measure of resolving power of eye



1Min of arc at nodal point of eye

- Two points can be visualised as 2 different point when there any is at 1min of are at nodal point of eye
- VA = 1/MAR (MAR-Minimal angle of resolution)

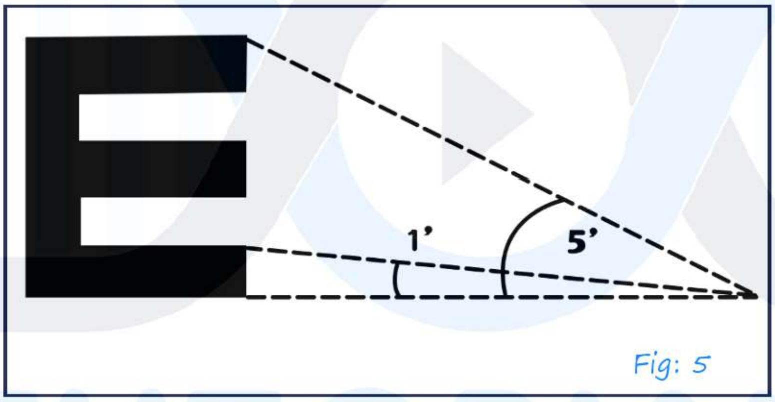
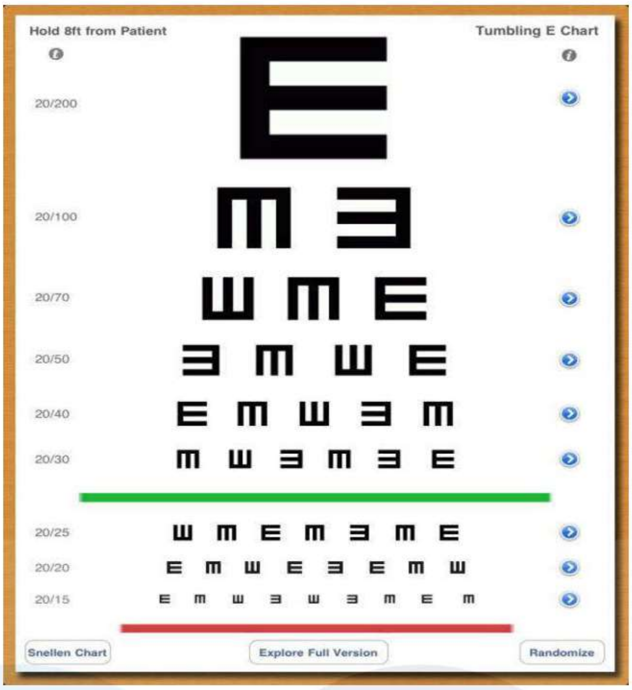
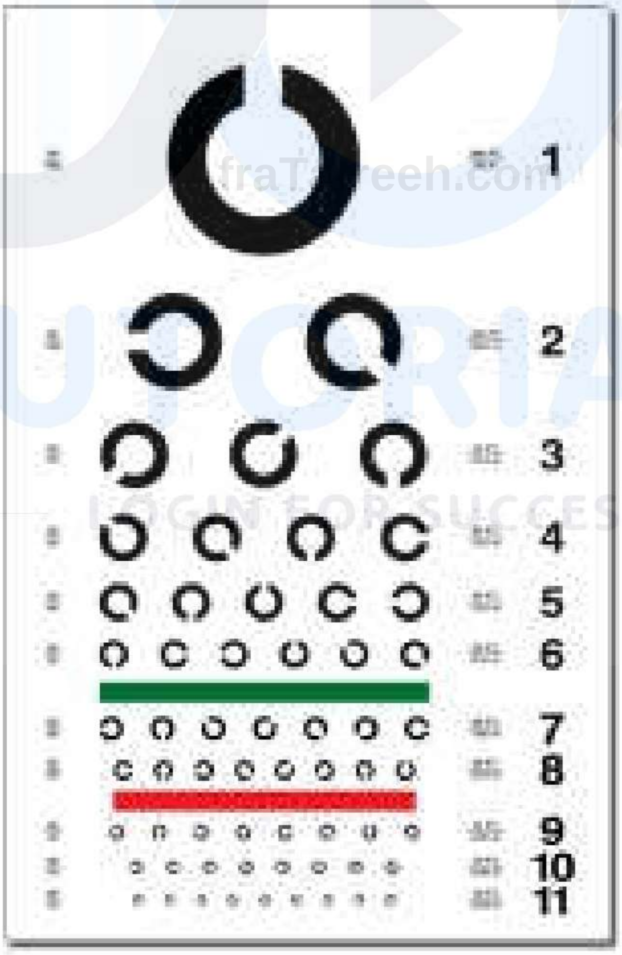


Fig: 5

- Consists of 5 individual details → 5min of arc
- Snellen’s chart is created in such a manner that each line makes an 5mm of are at its specific distance
- MAR is inverse of visual acuity
- for 6/60 vision what is MAR = 10 min of arc (60/6)
- for vision 6/60 angle subtended is - who is at 6mtr = 50 min
- if vision is 6/24 - angle subtended at 6mtr
- 24/6 x 5 = 20 min


Hold 8ft from Patient
Tumbling E Chart
20/200
20/100
20/70
20/50
20/40
20/30
20/25
20/20
20/15
Snellen Chart
Explore Full Version
Randomize

Tumbling E or charts Fig: 6


1
2
3
4
5
6
7
8
9
10
11

UC chart Fig: 7

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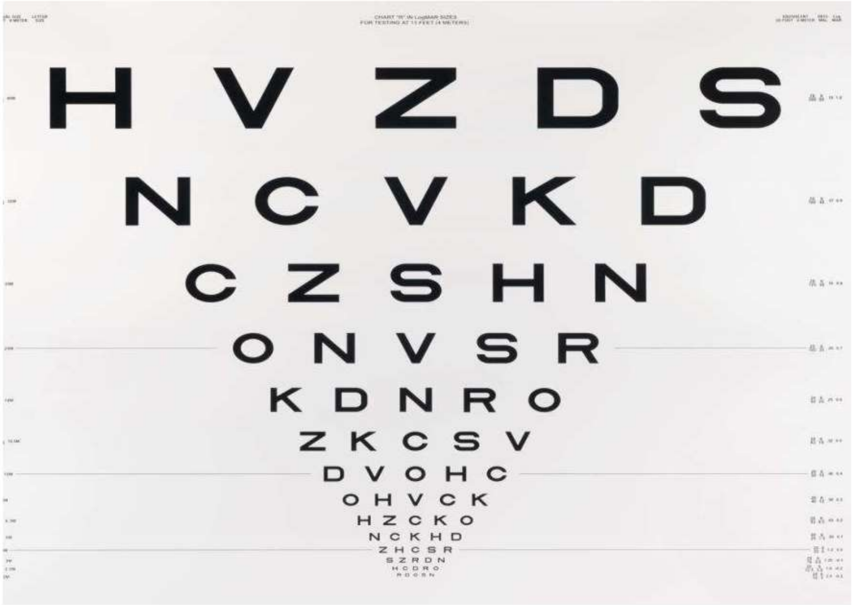
H V Z D S
N C V K D
C Z S H N
O N V S R
K D N R O
Z K C S V
D V O H C
O H V C K
H Z C K O
N C K H D
Z H C S R
S Z R D N
H C D R C
R C C S N

ETDRS chart or Log MAR chart

Fig: 8

- Early treatment diabetic retinopathy study
- Distance between 2letters is same as width of letter
- Distance between 2lines is same as height of letter
- Each letter has a value of 0.2 and line 0.2x5 =1log
- Useful for research purposes

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Infants 00:26:27

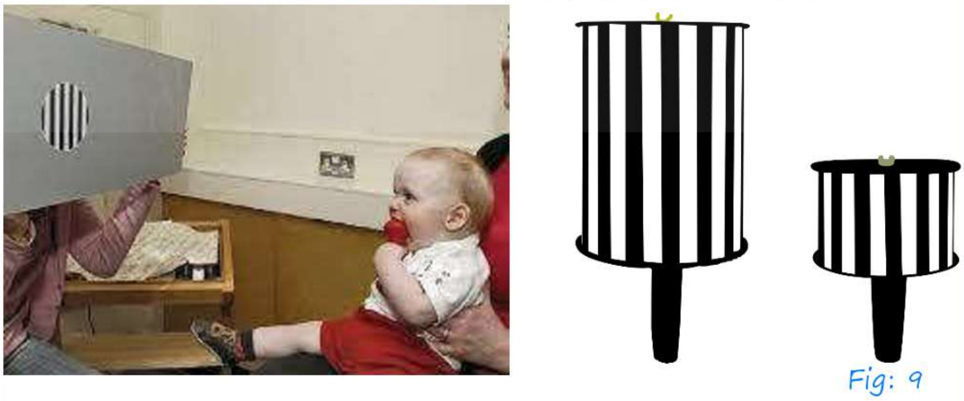


Fig: 9

- Visual evoked potential

  - Tests the integrity of visual pathway → Basically EEG recording
  - Recording of visual impulses which will be picked up by retina going to visual cortex
- OKN – Optokinetic nystagmus
  - Normal individuals can rotate eye to the movements of optokinetic drum
- PLT – Preferential looking test → Deviated to preferential things

Teller activity test

- Thickness of lines decreases

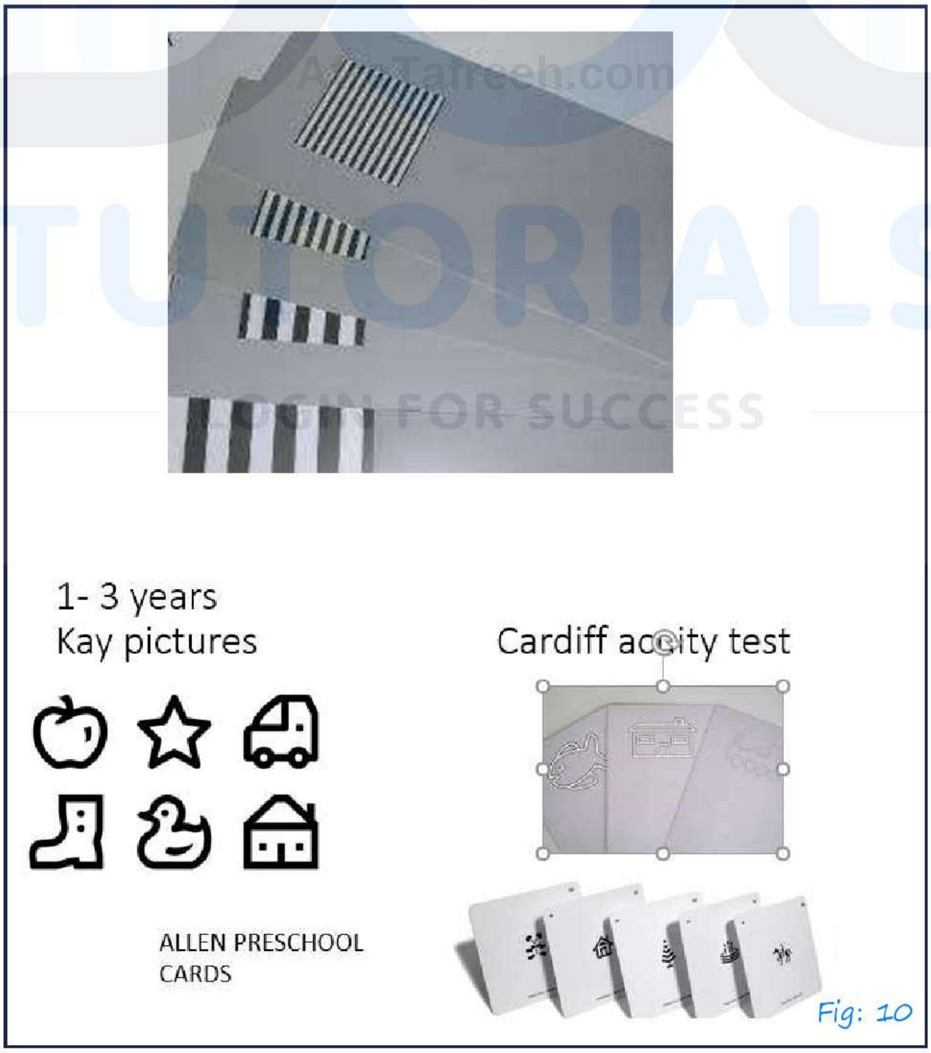
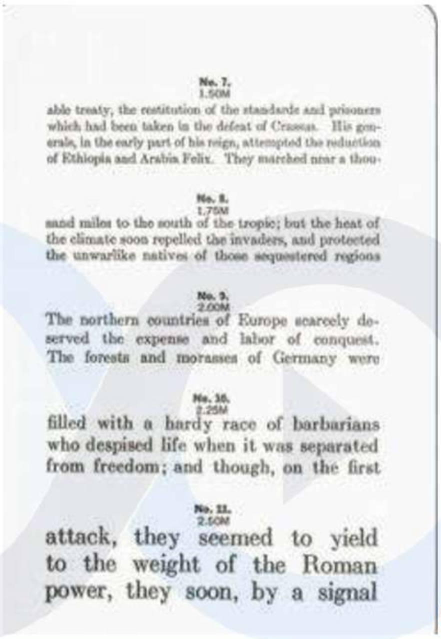

1- 3 years
Kay pictures
Cardiff acuity test
ALLEN PRESCHOOL CARDS

Fig: 10

- STYCAR – Screening test for young children and retards
- Based on persuit movements
- E chart → Can be used to children who can identify letters



No. 7.
1.50M

able treaty, the restitution of the standards and prisoners which had been taken in the defeat of Crassus. His generals, in the early part of his reign, attempted the reduction of Ethiopia and Arabia Felix. They marched near a thou-

No. 8.
1.75M

sand miles to the south of the tropic; but the heat of the climate soon repelled the invaders, and protected the unwarlike natives of those sequestered regions

No. 9.
2.00M

The northern countries of Europe scarcely deserved the expense and labor of conquest. The forests and morasses of Germany were

No. 10.
2.25M

filled with a hardy race of barbarians who despised life when it was separated from freedom; and though, on the first

No. 11.
2.50M

attack, they seemed to yield to the weight of the Roman power, they soon, by a signal

Fig: 11

- Jaeger near vision chart
- Checked at distance of 30-35cm
- Can read top most line → N/6

Fig: 12

- Hole is 1mm-ideally to be 1.2mm
- 6/60
- With pin hole
  - Vision improves- 6/9 or 6/6
  - Vision doesn't improve/worsens
- In myopia rays of light are focussed in front of retina → Large blur circle at retina

↓

Pin hole allows only single ray of light to go through aperture → Focused better towards retina and blur circle (Decreased)

- Lesion at macula → because of pin hole not allowing all hight rays to go in, central rays only go → Vision declines/Worsens
- Pin hole increases depth of focus

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Refraction 00:40:43

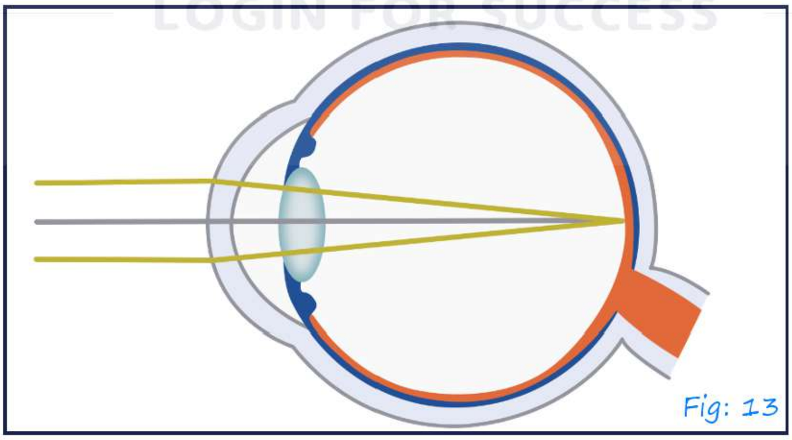

Fig: 13

- Emmetropia – Refractive status of eye when parallel rays of light are focused at 1point on retina → Unaccommodated state

- Ametropia – When parallel rays of light not focused on fovea
- Anisometropia – Different refractive status of 2 eye
- Anisokonia – Different size of image formed by eye

-difference is >2.5D

↓

Significant anisokonia

1D of spectacles → Magnification/minification by 2%

up to 5% = 2.5D

Lens 00:46:23

- Plus, lens – Black
- Minus lens – Red
- Convex lens
  - Thicker in centre
  - Image moves opposite to movement of lens
  - Magnification
- Concave
  - Thin in centre
  - Image moves in same direction of lens
  - Minifying lens
- Cylindrical lens
  - Correct astigmatism
  - power acts perpendicular to astigmatism
- Toric lens
  - Combination of sphero – Cylinder

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REFRACTIVE ERRORS

Myopia 00:00:10

- Also called as near sightedness
- Refractive power is more (>+60)
- Able to converge the rays earlier.

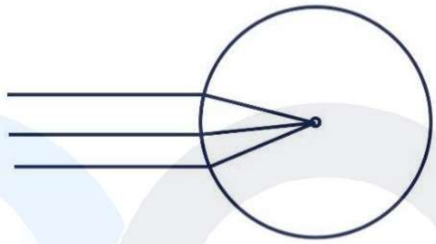

Fig: 1: Rays are converged in front of the retina

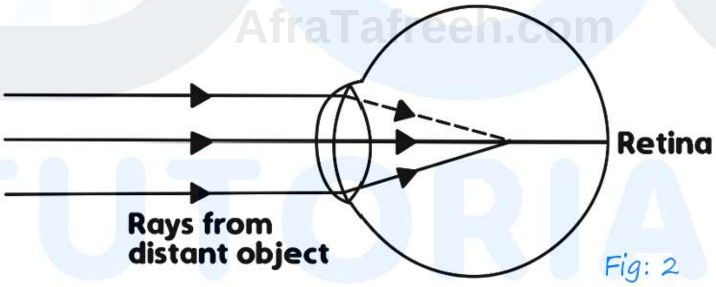

Fig: 2

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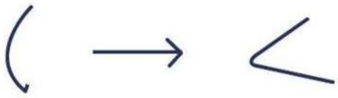
Types

I. **High axial length → axial myopia (most common)**

- Axial length, more than >24 mm E.g., Buphthalmos
- If my axial length = 24mm → + 60 D → spectacles → No need
- If my axial length = 25mm → + 63 D → spectacles' power → - 3D
- **1 mm increase in length increases power by ↑ 3D**

- If axial length is 26mm → spectacular power is -6D → Definition of pathological myopia (Axial length of >26 mm, Spectacles > -6 D, Posterior Segment Changes)

II. Curvatural myopia



- Increases in curvature of cornea → Decreases in radius of curvature
- 1mm decreases in radius of curvature → 6D increases ref. power

  E.g., Keratoconus/globus

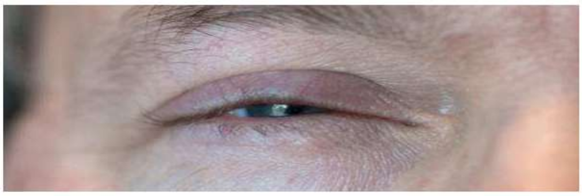
III. Positional myopia

- Anterior subluxation of lens leads to positional myopia
- Increased power of the eye – Image focused before retina

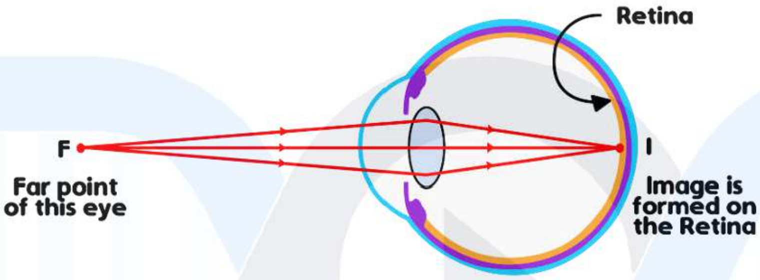
IV. Index Myopia

- Nuclear sclerosis/cataract
- Changes in the lens lead to this

Symptoms 00:09:47



Shut eye – Children come close and do this in order to see clearly

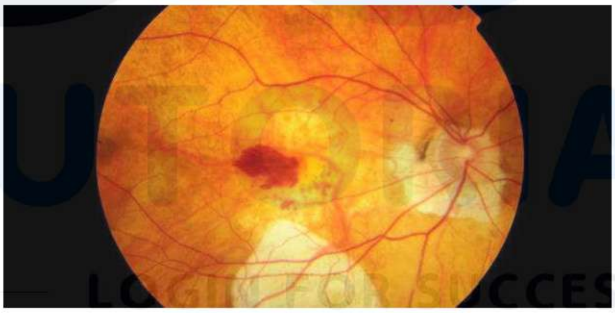

Retina
F
Far point of this eye
I
Image is formed on the Retina

(b) The far point (F) of a myopic eye is less than infinity Fig: 3

- Far point comes near (less than infinity)
- Does not accommodate for near objects → convergence insufficiency resulting in exophoria
- Large Eyeball, Large Cornea and Lens and **Deep AC**
- Higher tendency for developing POAG, pigmentary glaucoma, nuclear cataract
- Low ocular rigidity

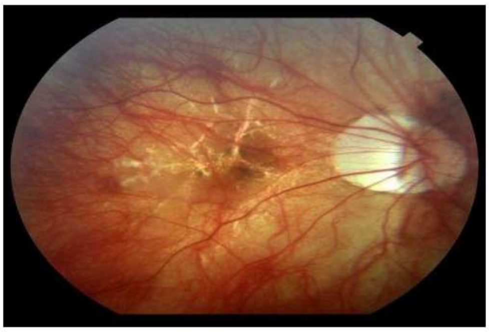
Features of myopia 00:14:50

- More frequency of degeneration like lattice, snow flake
- More chances of retinal detachments (rhegmatogenous)
- Choroidal hemorrhage in macula: **foster Fuch's spot**
- **Lacquer cracks**: breaks in Bruch's membrane
- **Myopic crescent**: disparity b/t sclera (globe enlarges) and RPE
- **Posterior staphyloma:** ectasia of sclera
- **Tigroid Appearance of Retina**
- Large disc, large disc cup ratio
- Visibility of choroidal vessels, RPE thinning
- Tilted disc: dragging of retina nasally over disc

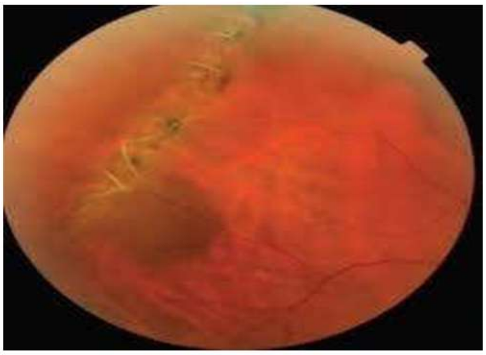


Myopic crescent

Foster Fuch's spot Fig: 4



Lacquer cracks Fig: 5



Criss cross @ retina lines

↓

Lattice degeneration

Fig: 6

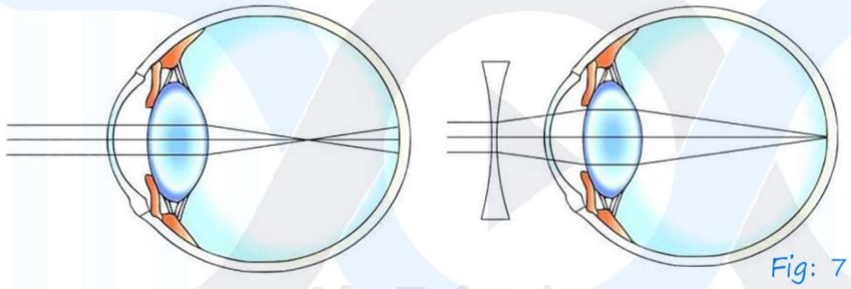


Fig: 7

- Correction of myopia
  - Diverging lens are to be given
  - Spectacles, Contact lens
  - Lasik can also be done
- Never overcorrect
  - Under correction has been believed to reduce the accommodative stimulus and demand at near and subsequently reduce the blur drive for accommodation increasing myopia
  - Fully corrected now, development of normal accommodation and convergence (atleast for children)

Hypermetropia 00:20:00

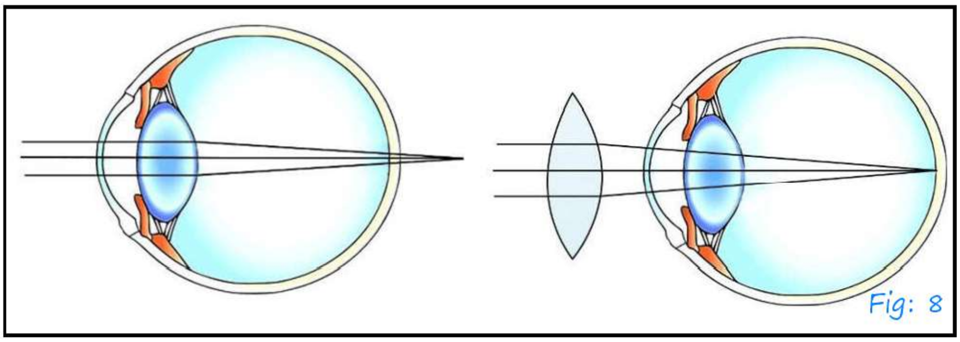


Fig: 8

- Long sightedness – Can't see near objects
- Converging power < +**60D**
- Image is focused behind retina
- Functional hypermetropia – in internal ophthalmoplegia → paralysis of intrinsic muscles of the eye → sphincter and ciliary muscle → loss of accommodation → hypermetropia
- Total = latent (exerted by the tone of ciliary muscle – abolished by cycloplegia) + Manifest (facultative: corrected by accommodative effort) + Absolute (uncorrected hypermetropia)

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Symptoms 00:23:00

- Asthenopia – tiredness, headache, irritability, brow pain

Types

I. Axial hypermetropia
   a. Axial length <24 mm
   b. E.g., Microphthalmos, nanophthalmos, Congenital rubella syndrome
   c. If axial length is 22mm → spectacles are supposed to be +6D

II. Curvatural hypermetropia
   a. Decreased curvature → increases radius of curvature
   b. e.g., Cornea plana (flattened cornea)
   c. 1mm decrease of curvature → +6D of refractive error

III. Positional hypermetropia
   a. Posterior displacement of lens

IV. Index
   a. Cortical cataract

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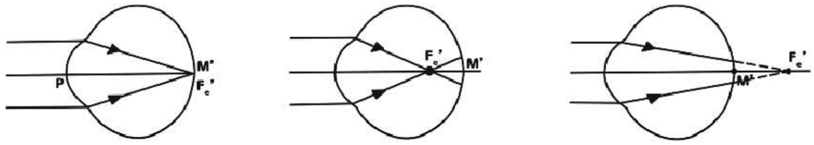
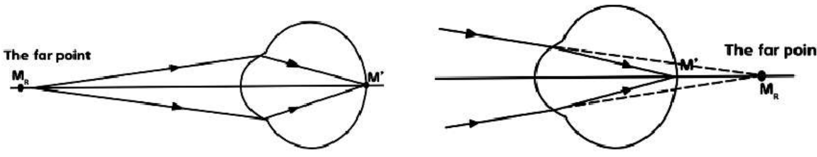


Left to right: unaccommodated emmetropia; myopia; and hyperopia



Far point in(left)an unaccommodated myopic eye and(right)an unaccommodated hypermetropic eye

Esophoria/convergence squint

Fig: 9

Treatment

i. Glasses → convex

ii. Contact lens

iii. LASIK

Astigmatism 00:28:37

- 2 or more than one point of focus at the retina
- Image is not sharp because of this

Types of Astigmatism

i. Regular astigmatism → vertical and horizontal meridian (perpendicular)

  a. All points on vertical meridian have same power and all points on horizontal meridian have same power.

ii. Irregular astigmatism

  a. Not perpendicular to each other

  b. Different powers at different sites.

  c. E.g., Corneal scar, Keratoconus (late cases), Advanced pterygium

Types of Regular Astigmatism 00:32:25

A. WTR

  a. With The Rule

  b. Predominant in the Population

c. Vertical meridian → More Convex → More Curved → Higher power

d. Horizontal meridian → Flatter → Lesser Power → More diameter

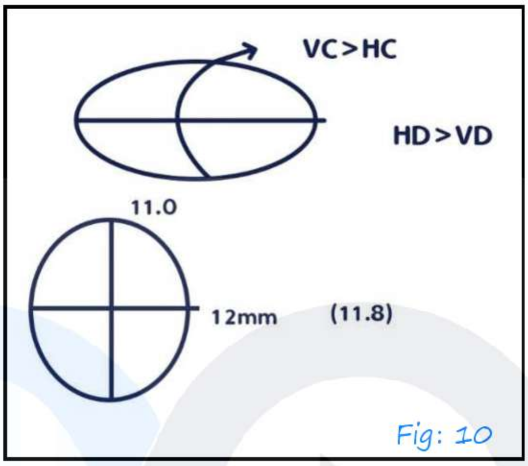


Fig: 10

B.ATR

a. Against the rule

b. Horizontal diameter is less than vertical diameter

c. Horizontal Curve is less than vertical curve

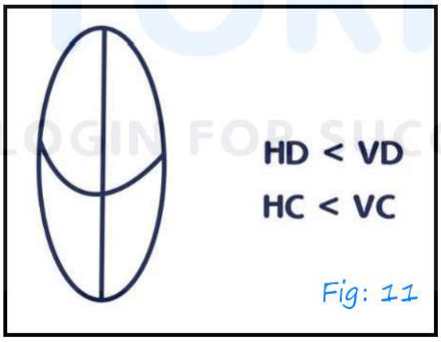


Fig: 11

C.Oblique

a. Both the meridians are perpendicular but not at 90° & 180°

Symptoms 00:37:38

- Suffer from maximum levels of asthenopia

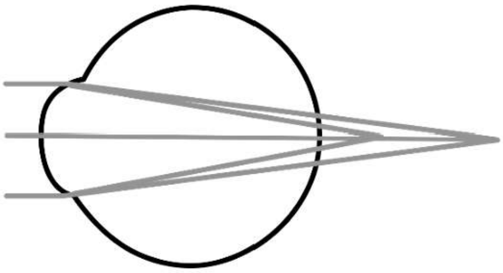


Fig: 12

Compound hypermetropic astigmatism

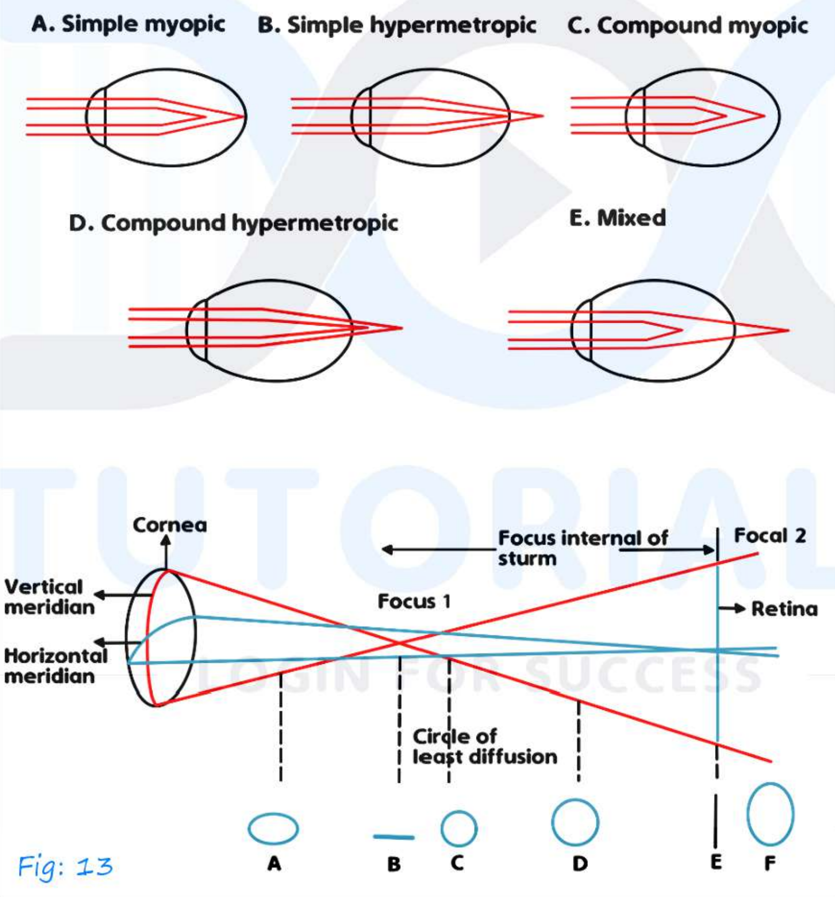

A. Simple myopic
B. Simple hypermetropic
C. Compound myopic
D. Compound hypermetropic
E. Mixed
Cornea
Vertical meridian
Horizontal meridian
Focus internal of sturm
Focal 2
Focus 1
Retina
Circle of least diffusion
A
B
C
D
E
F

Fig: 13

Strum's Conoid

- Configuration of rays
- Vertical rays of light are focused earlier and horizontal rays of light are focused late
- If the light focusses at point A – Horizontal oval

- If the light focusses at point B – horizontal line
- If the light focusses at C – sharp circle → Divergence of horizontal rays is equal to convergence of vertical rays
- If the light focusses at D – similarly Circular, but not the best
- If the light focusses at E – Vertical line
- If the light focusses at F – Vertical oval

a. If retina @ B → simple hypermetropic (SHA) astigmatism

b. If retina @ E → simple myopic astigmatism (SMA)

c. If retina @ C → mixed astigmatism (Sharpest Image – Circle of least diffusion)

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Note:

The distance between where the vertical rays and the horizontal rays converge is called as the Focal Interval of Sturm

Prescription 00:47:09

- –2.5 DS (diopter sphere) → myopia
- +2.5 DS → hypermetropia
- –2.5 DC @ 90° (diopter cylinder) → SMA
- +2.5 DC @90° → Simple hypermetropic astigmatism

- +2.5 DS/+1 DC @ 90^0 → Compound hypermetropic astigmatism
- -2.5 DS/-1 DC @ 90^0 → Compound myopic astigmatism
- +1 DC/-1 DC @ 90^0 → Mixed Astigmatism

Treatment

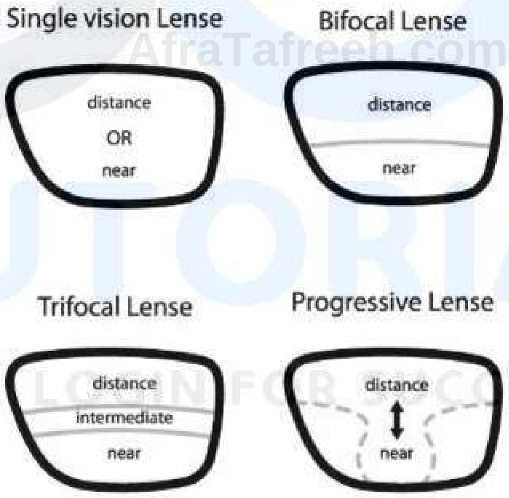
- Cylindrical/Toric lens
- Astigmatic LASIK

PRESBYOPIA AND APHAKIA

Presbyopia 00:00:19

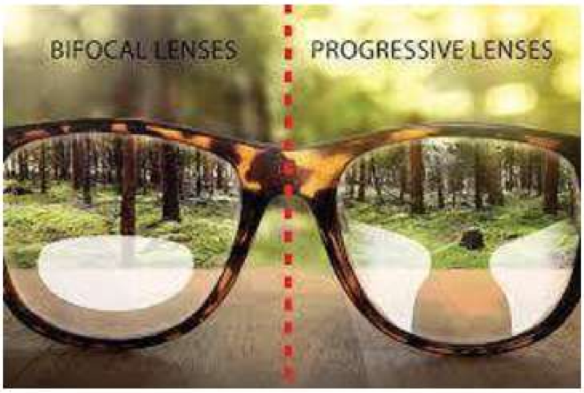
- Age related degenerative condition
- Ciliary muscles are weak and lens become rigid

Fig: 1



Single vision Lense
distance
OR
near
Bifocal Lense
distance
near
Trifocal Lense
distance
intermediate
near
Progressive Lense
distance
near

Fig: 2



BIFOCAL LENSES
PROGRESSIVE LENSES

Fig: 3

- Bifocal lens → lower segment for near segment

Accommodation 00:04:42

- Range of accommodation
- Distance between far (infinity) and near point (30cm)

Power of accommodation

- Refractive power of eye in fully accommodative state - power @ rest = power to focus @ near – power to focus @ far
- 1D= 1/distance in meter

  $$\text{power} = \frac{1}{\text{Focal length}}$$

Remember

- Emmetropia for point infinite
- Myopia far point – in front of eye so + sign
- Hypermetropia → far point behind the eyeball (Virtual so -sing)
- Child emmetropic of 12-year-old near point 10cm so, amplitude of accommodation is 1/0.1 – 1/infinity = 10 D
- Calculate amplitude of accommodation where patient is myopia - 2D near point 10cm 2D is already given as power to focus at far, so amplitude will be 1/0.1 -2 = 10-2 = 8D
- For Hypermetropes → 10+ 2= 12D

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Aphakia 00:09:04

- Absence of lens
- Most common cause – post surgery
- Other causes – Trauma
  - Congenital
- Lens contribute → +15 to + 17 D

↓

If removed

↓

High Hypermetropia

Clinical features

- Poor vision
- Jet black pupil
- Iridodesis- Tremulousness of iris
- Deep anterior chamber
- 2 purkinjee image →

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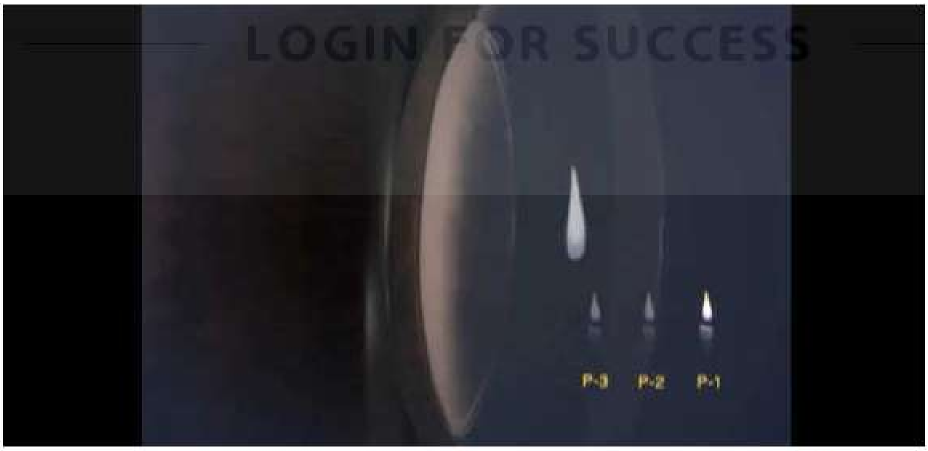


Fig: 4

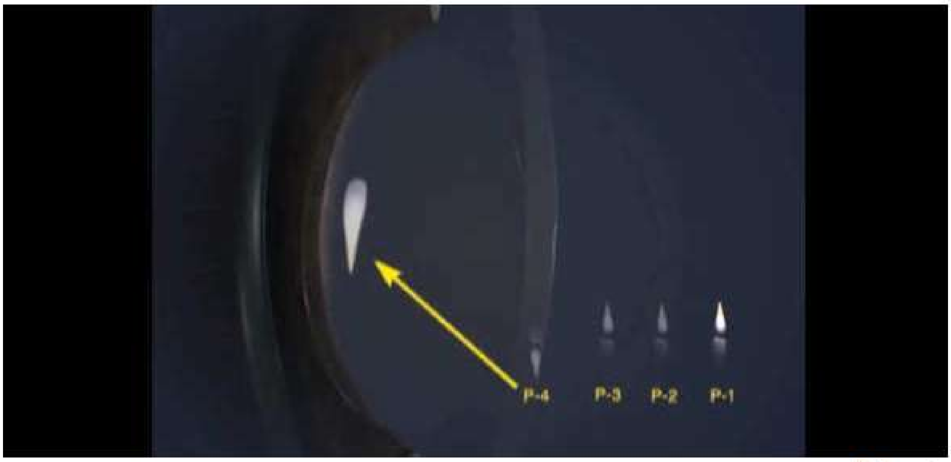


Fig: 5

↓

Also called catopteric image

- 1st image – anterior surface of cornea
- 2nd image - posterior surface of cornea
- 3rd image – anterior surface of lens
- 4th image – posterior surface of lens (concave surface

↓

inverted)

Treatment

- Best is to place corrected IOL
- Contact lens → Minimal magnification of images
- Aphakic glasses → + 15 D

↓

30% Magnification

α → not tolerable

→ Ideal power +10D

Problems

- Frog eye

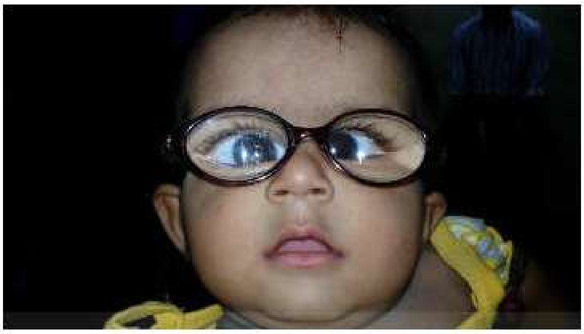


Fig: 6

- Pin cushion distortion

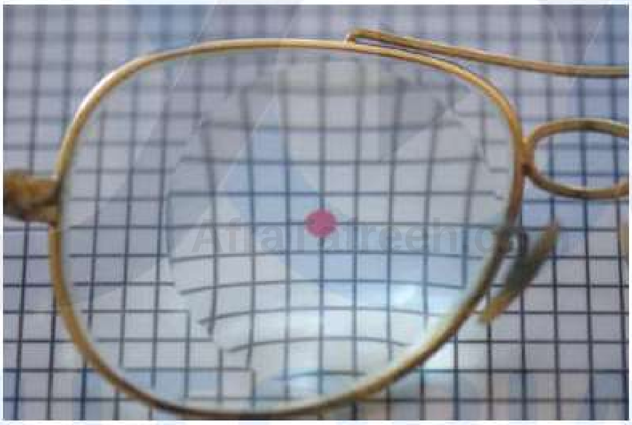
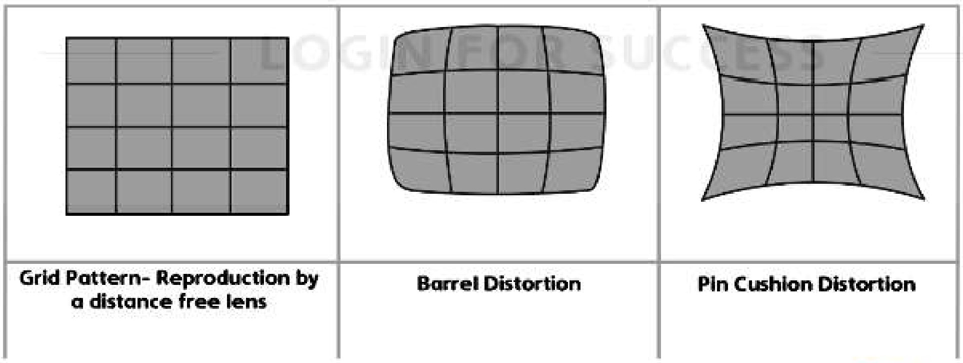


Fig: 7



Grid Pattern- Reproduction by a distance free lens | **Barrel Distortion** | **Pin Cushion Distortion**

Fig: 8

- Barrer → Concave
- Jack in box phenomenon

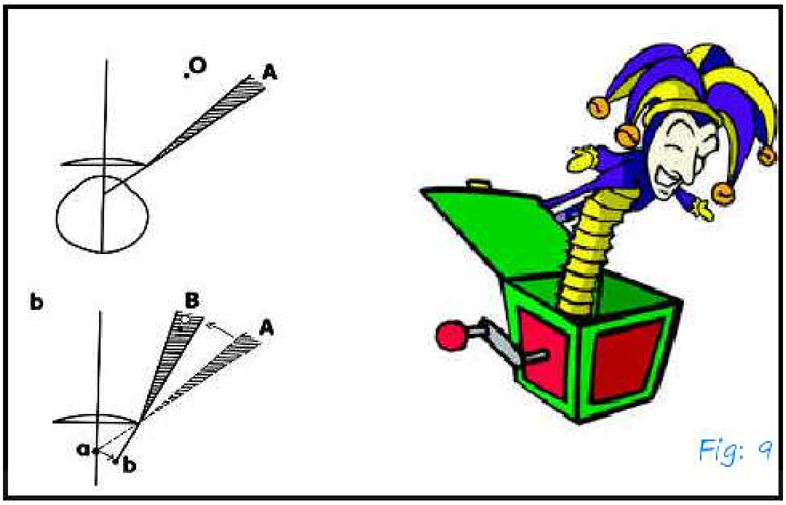


Fig: 9

- Because of high plus power, periphery behaves like prism

  ↓

  Because of edge effect → there is a creation of area which is scotoma

  ↓

  As we move eye towards objects it is not visible as it comes an area of scotoma

  ↓

  Close stop moves away → it can see the object → Jack and box phenomenon

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Pseudophakia 00:20:22

- Presence of IOL
- 3 purkinjee image +

Contact lens 00:20:52

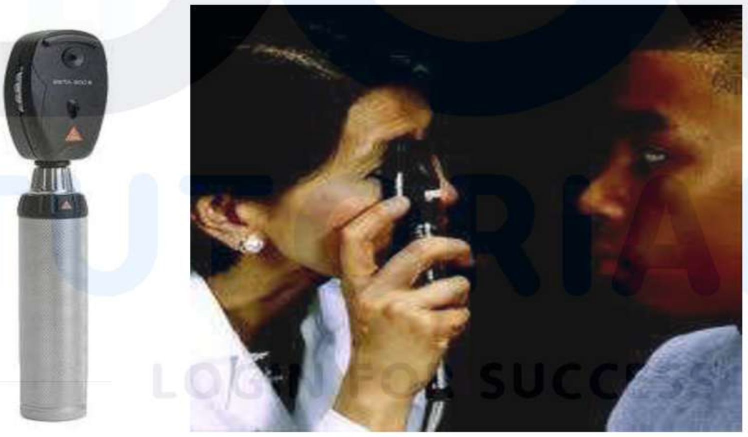
- Hard – PMMA, poor oxygen delivery
- Soft- HEMA (Hydroxy ethyl meth acrylate)
  (Most common used)
- RGP- Rigid gas permeable → copolymer of PMMA and silicone, good O_2 delivery

OPHTHALMOSCOPE AND RETINOSCOPE

Ophthalmoscope 00:00:12

Tabular column

Direct Ophthalmoscope	Indirect Ophthalmoscope
routine central fundus 10-degree fundus seen only	Centre + periphery 30 degree (8 DD fundus) seen stereopsis 3D view
image virtual, erect, high magnification 15 times 2 D image	image real, inverted, less magnification only 5 times
no view of retina when media not clear	retina seen even if media not clear



Distant direct ophthalmoscopy Fig: 1

direct ophthalmoscope

↓

Distant direct ophthalmoscope

↓

Placed at a distance of 25 mm

↓

The moment you place, we will see a red glow

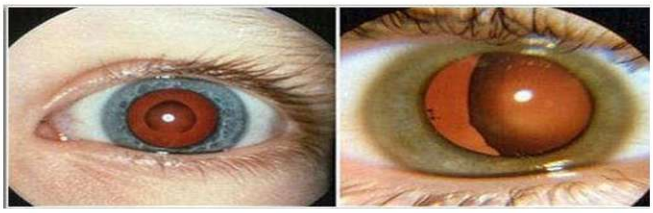


Fig: 2

- Black areas → media opacity
- Edge of lens seen → lens subluxated

Fig: 3

TC. Explanation.

Direct ophthalmoscope

- Disc – when patient looks straight
- Macula – when patient looks into light of ophthalmoscope
- Indirect ophthalmoscope

↓

Placed at arm's distance

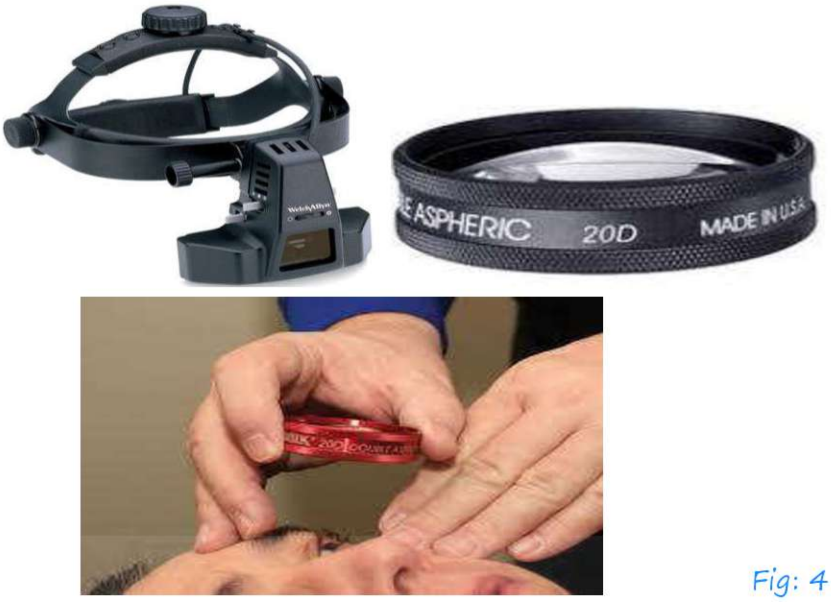

ASPHERIC
20D
MADE IN U.S.A.

Fig: 4

Retinoscopy 00:07:11

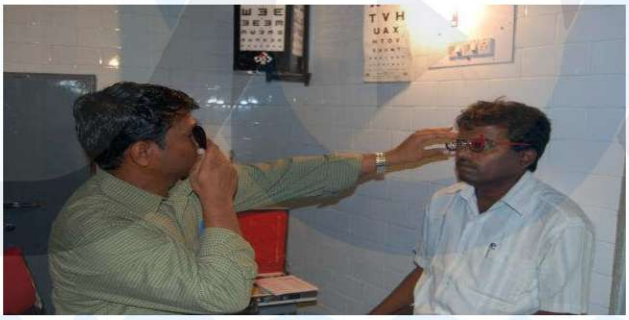
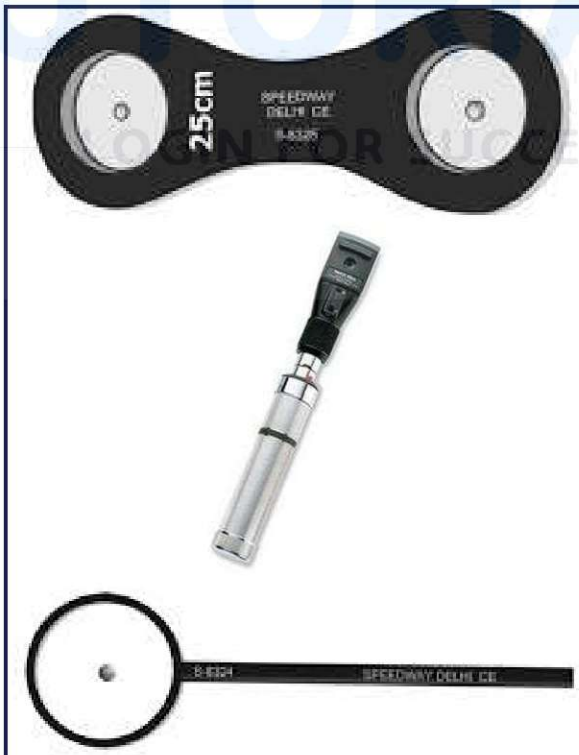


Fig: 5

Fig: 6


25cm
SPEEDWAY DELHI CE
SPEEDWAY DELHI CE

Priestly smith retinoscope

↓

Not used now a day

Retinoscope

↓

Most common used

- Retinoscopy
  - Find out refractive status of a patient
  - Based on principle of neutralization
  - Retinoscopy reflex

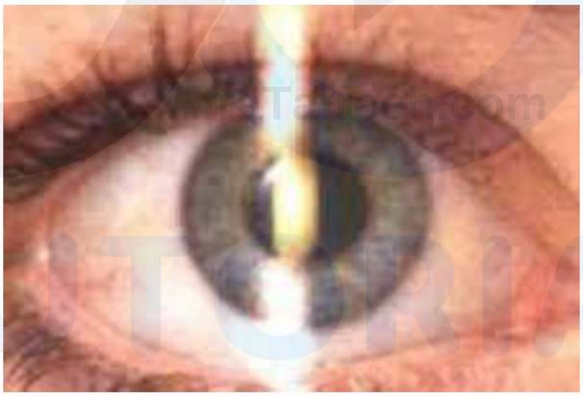


Fig: 7

Prerequisites of retinoscopy

1.Dark room

2.Placed at a distance of 1m/arm distance 75 cm

3.Cycloplegic agents

- Light from retinoscope thrown on patient's eye, this light goes on patient's retina, depending upon the refractive power of patient, the light is reflected in pupillary region.

- When the retinoscope is moved horizontally, reflex is also moved
- Trial frame / lens are added for neutralization

Fig: 8

- Why 1 meter distance → for convenience
  - For 1 meter correction of 1D
  - For 0.75-meter correction – 1.5D
- Cycloplegics
  - Children < 3 years –
- Atropine strongest, longest acting (2 weeks)
- Atropine ointment applied

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- Retinoscopy done 03 days after application of atropine ointment
- Ointment has lesser systemic absorption
  - Atropine side effects – fever, rash
  - Children >5 yr - <10 yr → cycloplegic – cyclopentolate

1-2 days action

+ Squint → atropine ointment

- >10yr -35 years → cyclopentolate / Homatropine

(action – 1 day)

Correction factors

- Atropine → - 1 D
- Cyclopentolate → - 0.75 D
- Homatropine → - 0.5 D

- When we move retinoscope horizontally, reflex also moves in same direction
  - Emmetropia
  - Hypermetropia
  - Myopia < -1 D
- When reflex moves in opposite direction

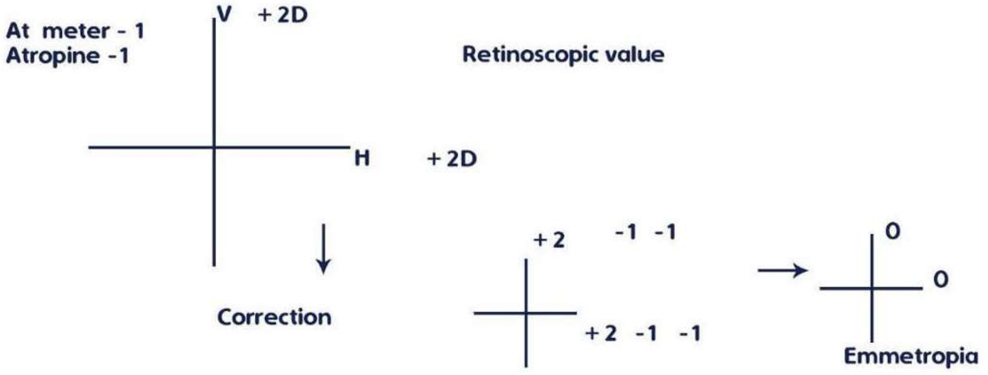
  ↓

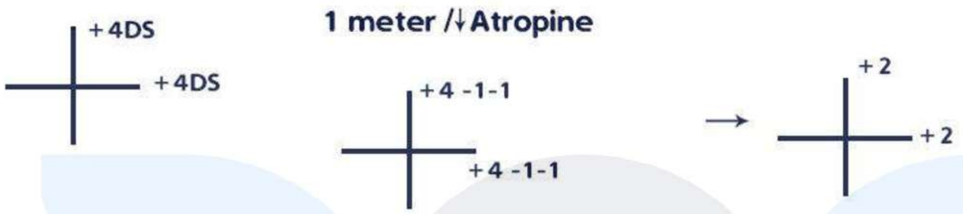
  Myopia >-1 D
- No movement

  ↓↓

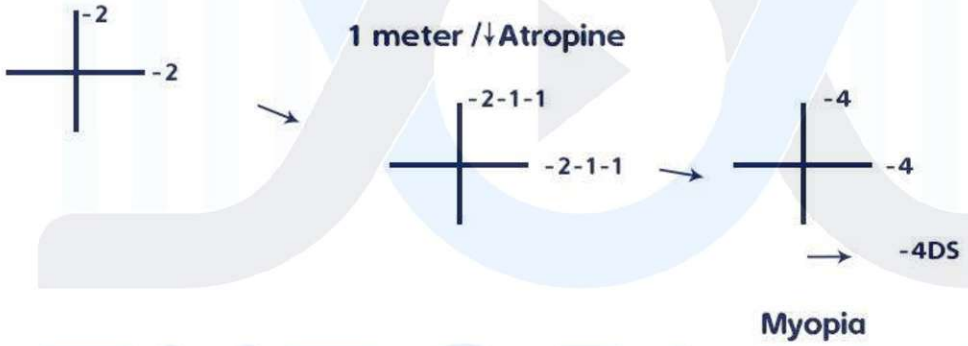
  Myopia = -1D

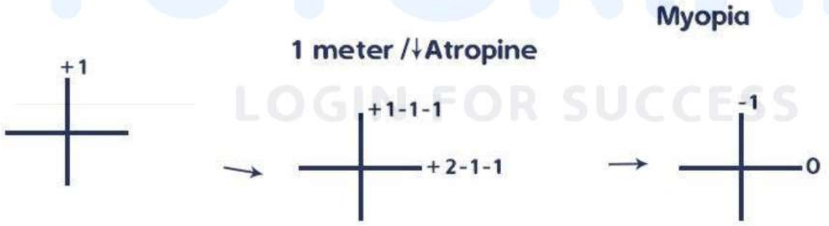
Power excess


At meter - 1
Atropine -1
V +2D
H +2D
Retinoscopic value
Correction
+2 -1 -1
+2 -1 -1
0
0
Emmetropia

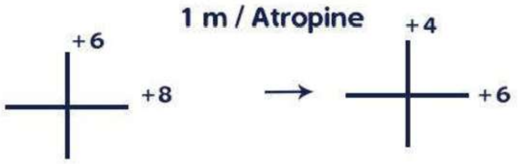

+4DS
+4DS
1 meter /↓Atropine
+4 -1-1
+4 -1-1
+2
+2

Power = +2DS ⟶ Hypermetropia


-2
-2
1 meter /↓Atropine
-2-1-1
-2-1-1
-4
-4
-4DS
Myopia


Myopia
1 meter /↓Atropine
+1
+1-1-1
+2-1-1
-1
0

Pres → -1D cylinder @180° ⟶ Astigmatism simple myopic astigmatism


1 m / Atropine
+6
+8
+4
+6

+4DS

want +2 more @ 180°

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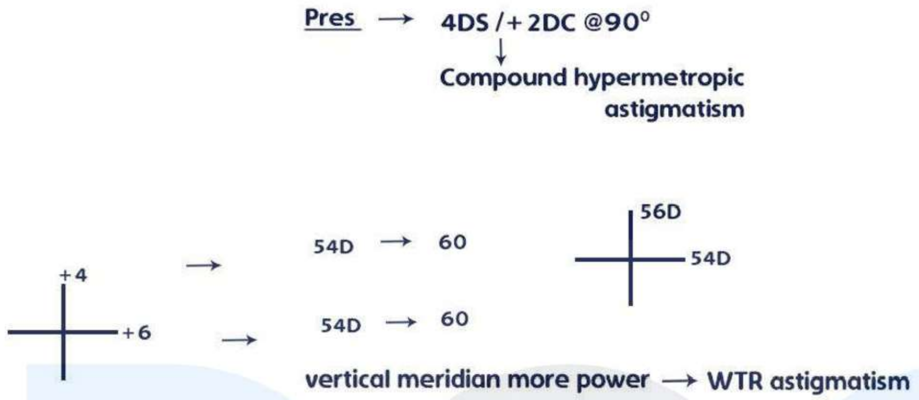
Pres → 4s + 4DS/+2DC @ 90°

↓

Compound hypermetropic astigmatism



Pres → 4DS /+ 2DC @90°

↓

Compound hypermetropic astigmatism

vertical meridian more power → WTR astigmatism

- + @ 90° → with the rule

  – @ 90° – against the rule

  – @ 180° – with the rule

  + @ 180 – against the rule

Q. What is glass prescription of the patient if retinoscopy is +3 D at both 90 and 180 at 1 meter and under atropine

A. + 1D

B. + 3D

C. +4 D

D. +5 D

Ans = -4Ds/-SDS 290°

Q. Transposed value of this prescription +3DS / 1.5 DC ay 90 degrees

A. +4.5DS / – 1.5 DC at 90 degree

B. +4.5 DS

C. +1.5DS / – 1.5 DC at 90 degree

D. +1.5DS / + 1.5 DC at 180 degree

Ans = +1.5DS / + 1.5 DC at 180 degree

↓

Compound hypermetropic astigmatism

- It implies transfer of lens power from one form to another so as to their meridian values remain the same in both the forms
- It means rewrite the expressions of its power without actually changing them.

1.SUM: The new spherical surface is given by adding algebraically the power of the sphere and cylinder

2.SIGN: Retain the power of the cylinder but change the sign.

3.AXIS: Rotate the axis of the cylinder through 90°

- Higher power of cylinder > sphere

  ↓

  Mixed astigmatism

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Example: + +1 DS / – 4 DC at 180 degrees

↓

+ 1 + (–4)

↓ mixed

– 3 DS / +4DC @ 90°

WTR

Q. Spherical equivalent of this prescription +3DS / + 2 DC at 90 degree is

A. +5DS

B. +4DS

C. +3DS /– 2DC at 180 degree

D. +4DS / – 2 DC at 180 degree

Ans = +4DS

1.Take half the value of the cylinder and

2.Add it to the sphere power.

In other words, as a formula the spherical equivalent spherical equivalent = sphere + (Cylinder)/2

Fig: 9

- Auto refractometers
- Horopters

  Trial frame

  ↓

  Hit and trial frame

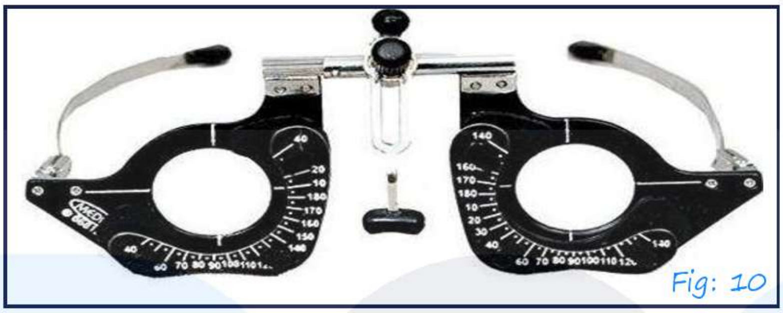

Fig: 10

Jackson cross cylinder

- Spherocylinder

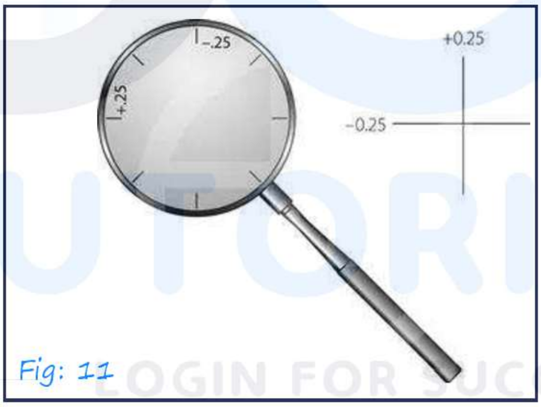

Fig: 11

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- Refinement of prescription
- To check accuracy
- Example: +0.5 DC @ 180
- Sphere power ½ of cylinder with opposite side
- Refinement of cylindrical valve
- Duochrome test

  ↓

  Refine my spherical power

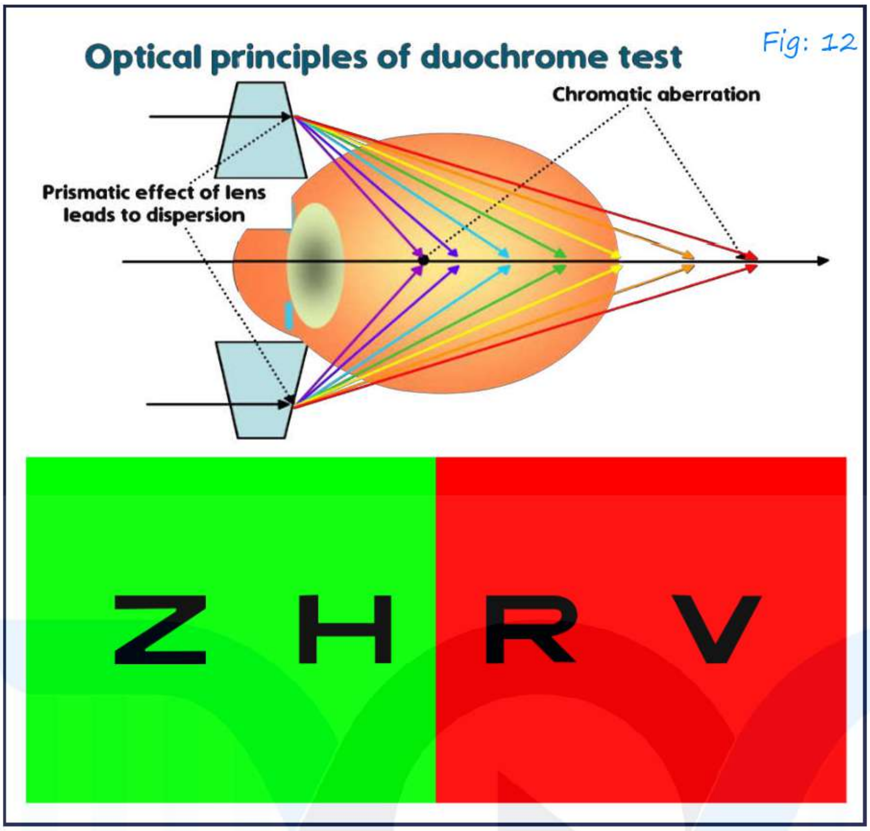
Optical principles of duochrome test
Chromatic aberration
Prismatic effect of lens leads to dispersion
Z H R V

Fig: 12

- Basically, a box with red and green area
- Based on optical aberration

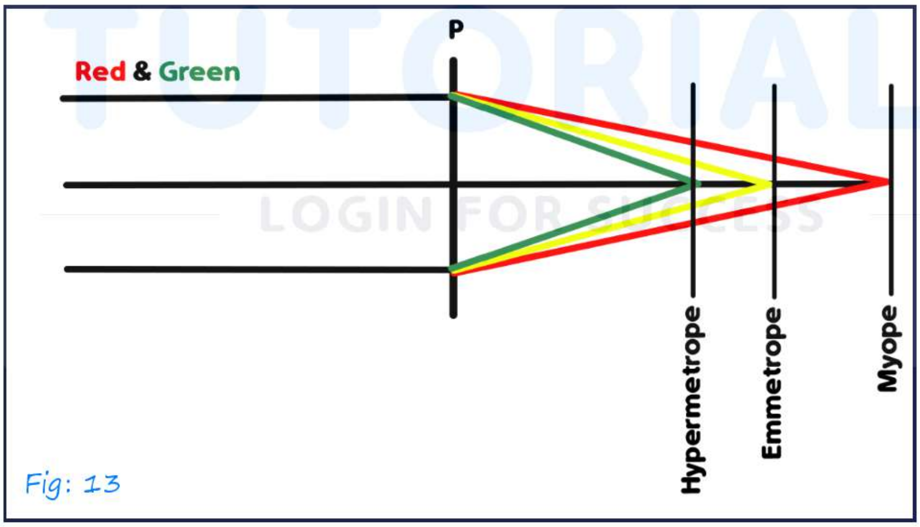
P
Red & Green
Hypermetrope
Emmetrope
Myope

Fig: 13

– Red better → Myopic

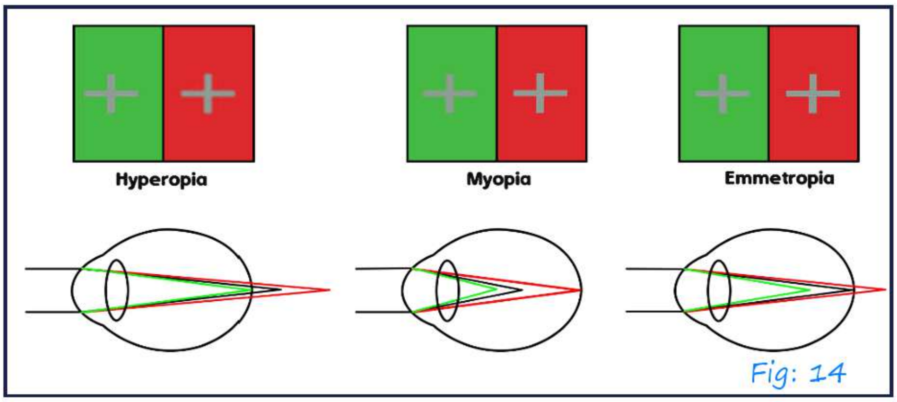
Hyperopia
Myopia
Emmetropia

Fig: 14

- Sees green color better – hypermetropic
- Sees red color – myopic
- Red and green equal – emmetropia
- If the letters of the green side are clearer + 0.25 D sphere is added
- If the letters on red side are clearer – 0.25D sphere is added

COMMUNITY OPHTHALMOLOGY

- Blindness → Best corrected visual activity <3/60 (WHO definition) in better eye or visual field <10 degree

Level	Presenting visual activity in better eye
- Mild vision impairment	- <6/12 but 6/18 or better
- Moderate vision impairment	- <6/18 but 6/60 or better
- Severe vision impairment	- <6/60 but 3/60 or better
- Blindness	- <3/60 and or a visual field of no more than 10° in radius around central fixation

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Causes of blindness/Visual impairment

- Most common cause of blindness – Cataract
- Most common cause of blindness in children – Vit A deficiency
- Most common cause of blindness in elderly → ARMD (Age related macular degeneration) (European countries)
- Most common infections cause of trachoma
- Most common cause of ocular morbidity – Refractive error

Vision 2020 Programme 00:03:40

- Vision 2020 programme
- Started in 1999 in Geneva
- Diseases targeted

  ↓

  T – Trachoma

  O – Onchocerciasis – river blindness

  R – Refractive error

  C – Cataract

  H – Childhood blindness
- In India , National programme for control of blindness and visual impairment (NPCB + VI) was launched in the year 1976 as a 100% centrally sponsored scheme
- Now 60:40 in all states and 90:10 in NE states with the goal of reducing the prevalence of blindness to 0.3% by 2020
- Under this program

  Q. minimal 21 million cataract surgeries were to be performed

  Q. cataract surgery rate: cataract surgery performed million population / year

  Q. Ratio of ophthalmologist 1:50000

Causes of blindness in India 00:06:41

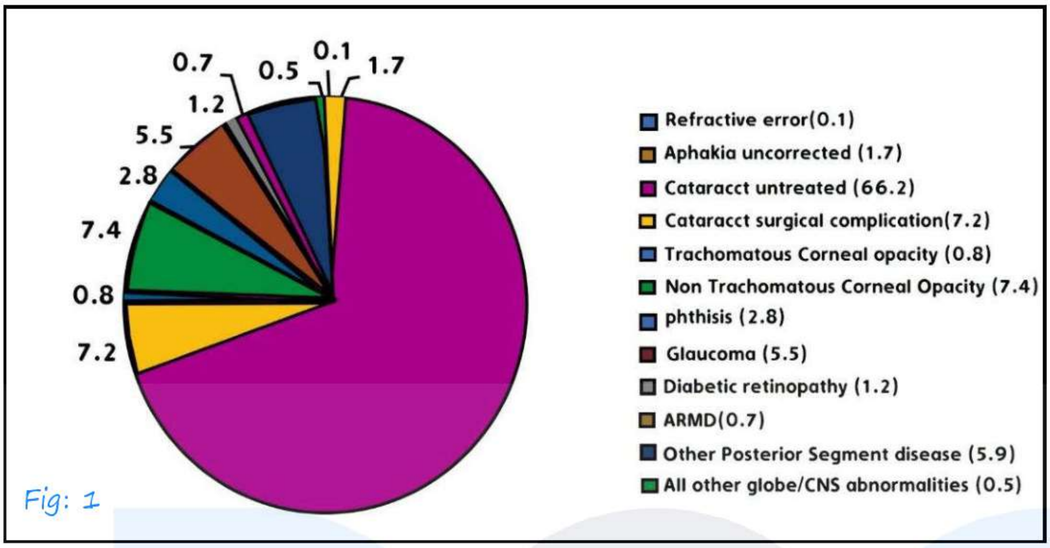

Fig: 1

- Most common cause – Cataract – 66%
- Second most common – cataract related complication (7.2)
- According to Indian vision 2020 programme, started in Goa in 2001

↓

Targeted diseases → Trachoma
Refractive error
Cataract
Childhood blindness
Corneal blindness
Diabetic retinopathy
Glaucoma

Blindness 00:08:44

- India → Presenting visual acuity <3/60 in better eye (or) VF <10°
- Social blindness: - Visual acuity (VA) <3/60 – better <1/60
- Economic blindness – VA <6/60 → presenting
- Legal blindness – VA <6/60 after correction
- Manifest blindness – VA < 1/60
- Absolute blindness – No PL (Perception of light)
- Almost 80% of blindness – avoidable

Curable → Cataract　　Preventable → Trachoma

- The national blindness and visual impairment survey 2015 – 2019 was conducted in order to provide the evidence about the present status of blindness and visual impairment in India
- The estimated prevalence of blindness was 1.99%

  SVI – 1.96%

  MVI – 9.81%
- Lowest in Thrissur Q
- Highest in bijnor Q

TUTORIALS LOGIN FOR SUCCESS

GET 2020 programme

- Started in 1997
- 8 Dec, 2017

  ↓

  Declared Trachoma eliminated in India

School eye health programme 00:14:09

- School teacher is trained to check the visual acuity of children 9-14 years
- Preferably female, science teacher
- 1:150 students
- If problem +, child would be referred

↓

Refraction would be checked

↓↓

Spectacles would be given free of cost

Rashtriya Bal Swasthya Karyakram 00:15:16

- To improve the overall quality of life of children enabling all children achieve their full potential
- Provide comprehensive care to all the children in the community
- For 4 D's → Defects of birth, diseases, deficiencies and developmental delays
- Early detection and free treatment and management, including surgeries at tertiary level
- 30 selected health conditions
- From birth till 18 years
- Eye conditions in RBSk

↓

1.Vit. A deficiency

2.Congenital cataract

3.ROP

4.Vision impairment

- National prophylaxis programme against nutritional blindness due to vitamin A deficiency (NPPNB due to VAD) was started in 1970 – with the specific aim of preventing nutritional blindness due to keratomalacia

Nutritional vitamin A prophylaxis programme

↓↓

Current scenario

- Clinical VAD has declined drastically during the last 40 years
  - There has been virtual disappearance of keratomalacia and a sharp decline in the prevalence of bitot spots
- Prophylactic vitamin A as per the following dosage schedule"
  - 100000 IU at 9 months with measles immunization.
  - 200000 IU at 16-18 months, with DPT booster
  - 200000 IU every 6 months, up to the age of 5 years.
  - Thus, a total of 9 mega doses are to be given from 9 months of age up to 5 years.

Infrastructure for vision 2020 00:17:10

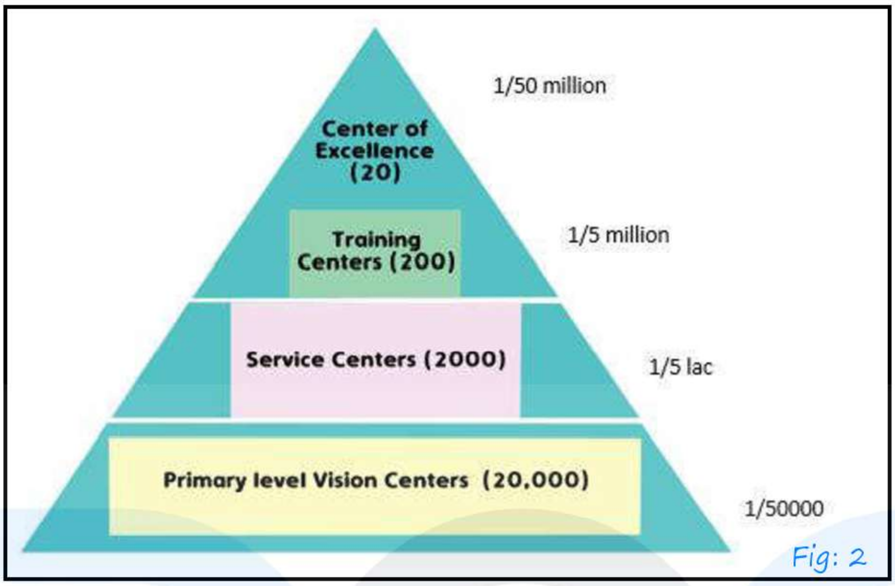
Center of Excellence (20)
1/50 million
Training Centers (200)
1/5 million
Service Centers (2000)
1/5 lac
Primary level Vision Centers (20,000)
1/50000

Fig: 2

Proposed Structure for Vision 2020: the right to sight

Tertiary	• Centers of excellence 20 – Professional leadership – Strategy development – Continued medical education (CME) – Lying of satadards and quality assurance – Research • Training center 200 – Tertiary eye care including retinal surgery corneal transplantation, glaucoma surgery etc.: – Training and CME
Secondary	• Services center 2000 – Cataract surgery

AfraTafreeh.com TUTORIALS LOGIN FOR SUCCESS

	- Other common eye surgeries - Facilities for refraction - Referral services
Primary	• Vision center 20000 - Refraction and prescription of glasses - Primary eye care - School eye screening programme - Screening and referral services

INTRODUCTION, AQUEOUS PRODUCTION & DRAINAGE

- Aqueous and vitreous humor contribute to intraocular pressure.
- Aqueous humor → 0.3ml → powerful determinant → continuous secretion

  Vitreous humor → 4 ml

Aqueous humor

- Plasma infiltrate
- Produced by 3 processes

  D – Diffusion

  U – Ultrafiltration

  S – Secretion → most important

  ↓ (70%)

  Active process
- Aqueous is produced by pars plicata → non pigmented epithelium
- Rate of secretion → 2.3 ml/min

Concentration of aqueous

- ATE more → pyruvate

  Lactate

  Ascorbate
- EAT less → protein

  Glucose

  Urea

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- Concentration of aqueous is different from plasma

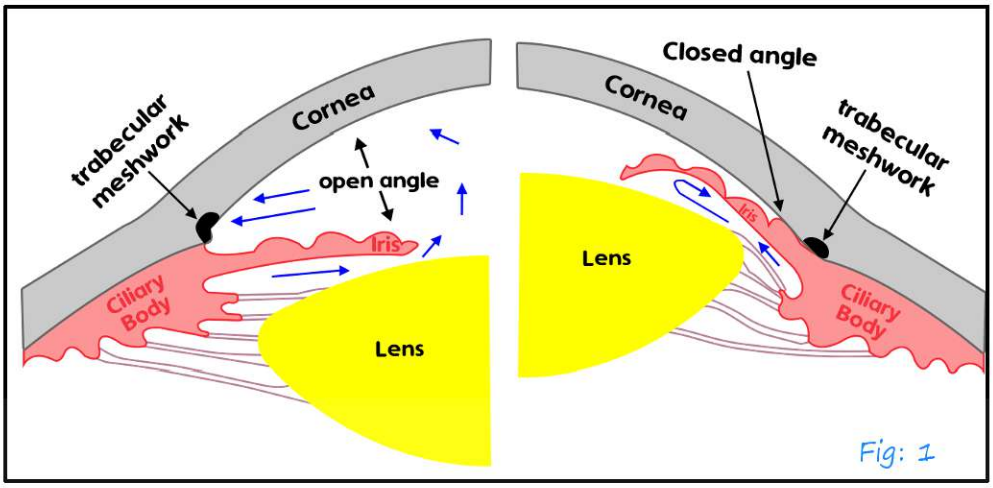

trabecular meshwork
Cornea
open angle
Iris
Ciliary Body
Lens
Closed angle
Cornea
trabecular meshwork
Iris
Lens
Ciliary Body

Fig: 1

Aqueous produced in ciliary body

↓

Posterior chamber

↓

Through pupil enters anterior chamber

↓

Then encounters through various structures

Trabecular outflow:

Trabecular meshwork

↓

Episcleral veins

↓

Collector channels

↓

Ophthalmic vein

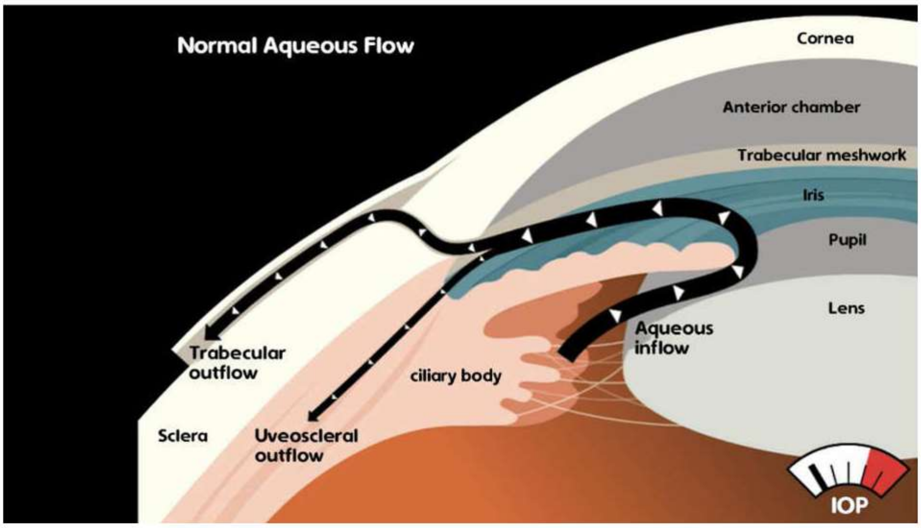

Normal Aqueous Flow
Cornea
Anterior chamber
Trabecular meshwork
Iris
Pupil
Lens
Aqueous inflow
ciliary body
Trabecular outflow
Sclera
Uveoscleral outflow
IOP

Fig: 2

- IOP independent pathway → unconventional pathway because less than 10% aqueous drains.

↓

Uveoscleral pathway

↓

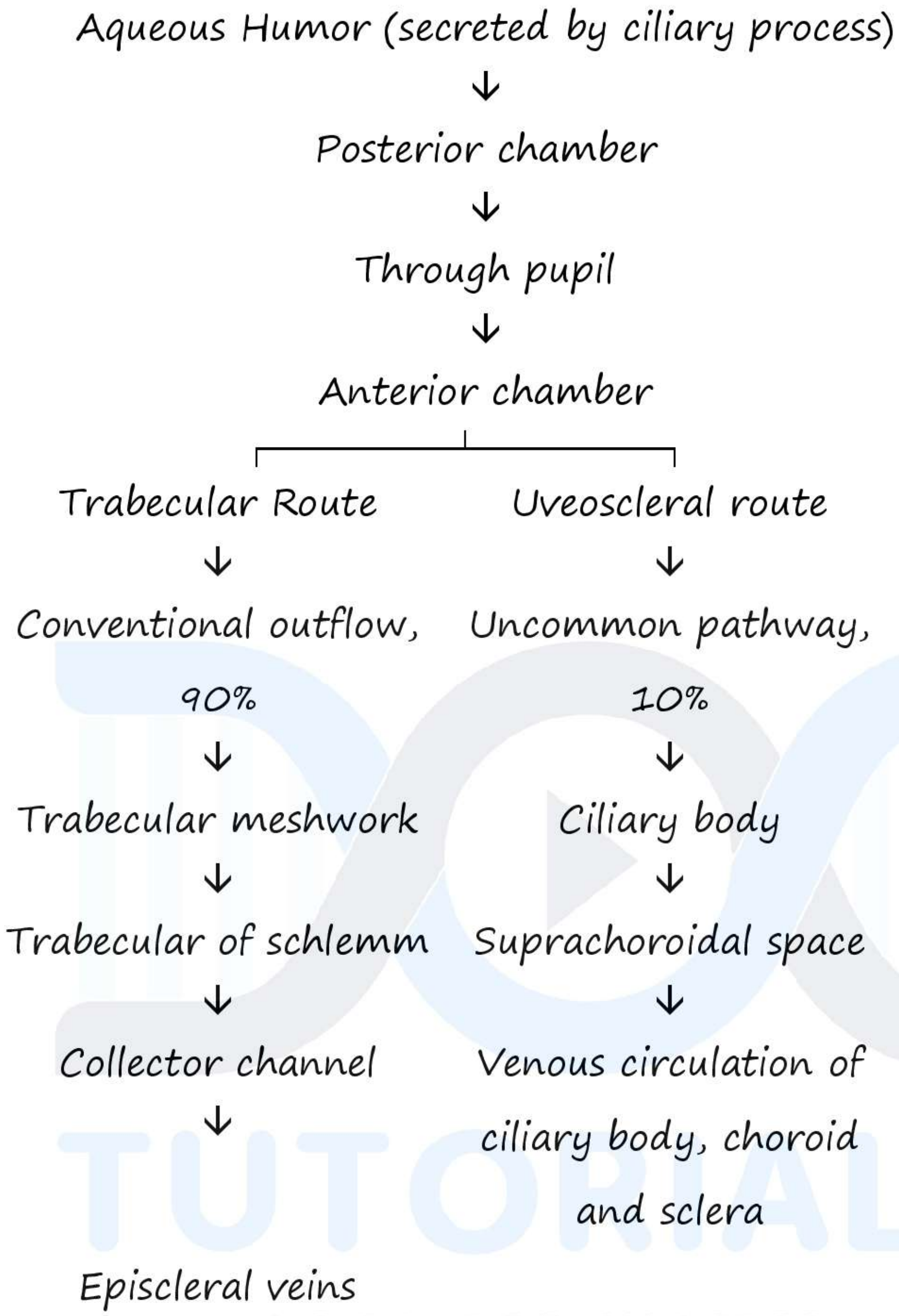
Absorbed by watson's plexus.

Drainage

i. Trabecular outflow → conventional

→ >90%

→ IOP dependent



Aqueous Humor (secreted by ciliary process)
↓
Posterior chamber
↓
Through pupil
↓
Anterior chamber

Trabecular Route	Uveoscleral route
↓	↓
Conventional outflow, 90%	Uncommon pathway, 10%
↓	↓
Trabecular meshwork	Ciliary body
↓	↓
Trabecular of schlemm	Suprachoroidal space
↓	↓
Collector channel	Venous circulation of ciliary body, choroid and sclera
↓	
Episcleral veins	

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- Episcleral vein → ophthalmic vein → cavernous sinus

ii. Uveoscleral outflow

- Unconventional
- <10%
- IOP independent pathway

Sclera → suprachoroidal spaces – vortex spaces

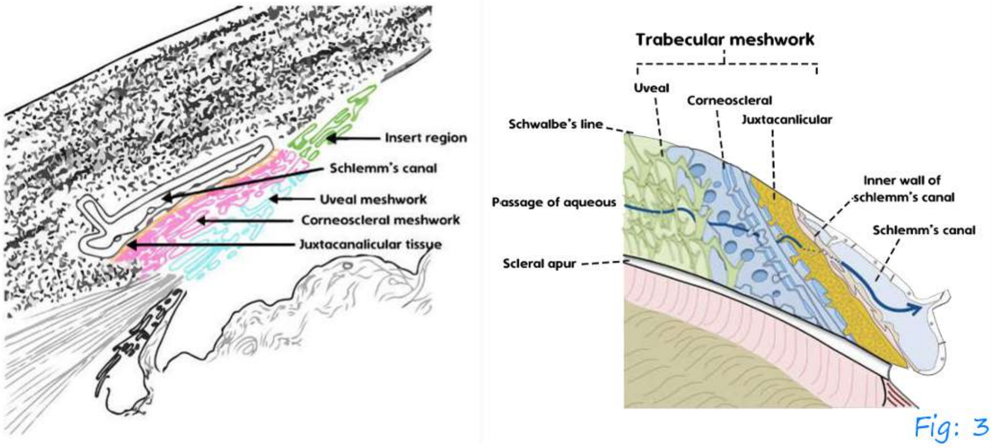
Insert region
Schlemm's canal
Uveal meshwork
Corneoscleral meshwork
Juxtacanalicular tissue
Trabecular meshwork
Uveal
Corneoscleral
Juxtacanlicular
Schwalbe's line
Passage of aqueous
Inner wall of schlemm's canal
Schlemm's canal
Scleral apur

Fig: 3

Trabecular meshwork

↓

- Outer one – uveal
- Middle → corneoscleral
- Inner most → Juxta canalicular / Inner wall of schlemms canal

↓

Smallest pore <10mm

↓

Maximum resistance to aqueous outflow

- Episcleral venous pressure: 6-10 mm Hg
- Tonography – facility of aqueous outflow

Determine by c value

C value = flow rate/IOP-EVP

C value is around 0.23 microlitres/min/mmHg

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ANGLE OF ANTERIOR CHAMBER ASSESSMENT & GONIOSCOPY

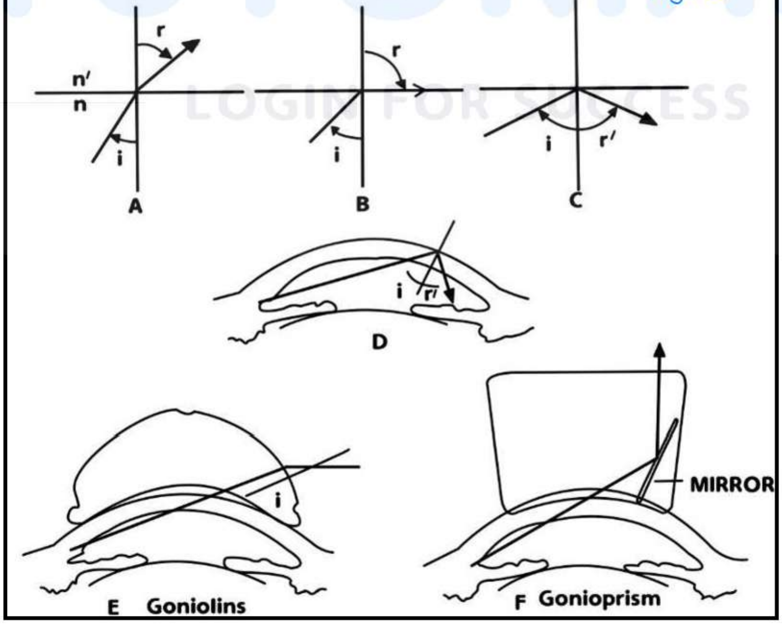
- Gonioscope – Instrument to study angle
- Total internal reflection → the rays of light from angle are going internally totally reflected
- To eliminate total internal reflection

  ↓

  Gonioscope is needed

Fig: 1

Fig: 2


r
n′
n
i
A
r
i
B
i
r′
C
i
r′
D
i
E Goniolins
MIRROR
F Gonioprism

- When the rays of light are moving from denser to rarer media → rays of light are refracted.
- Angle of incidence in denser media where the angle of refraction is 90 – critical angle
- Critical angle for cornea and air interface

  ↓

  46°
- If rays of light further increases

  ↓

  Total internal reflection
- Goniotons → Direct gonioscope

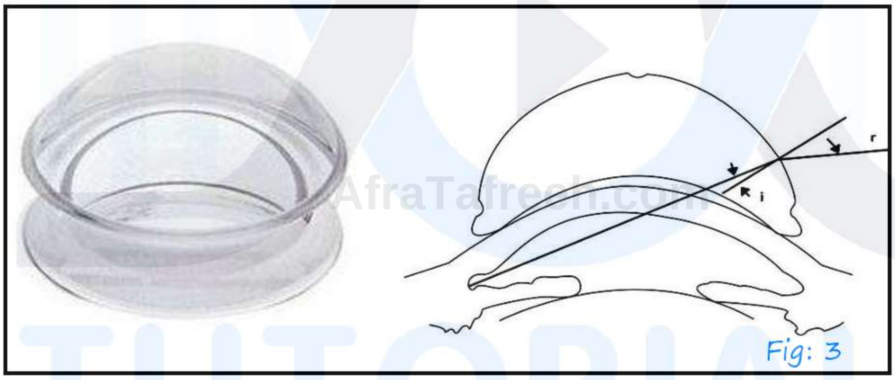


Fig: 3

↓

Prototype → Koeppe goniolens

↓

Rays of light are refracted to the angle and have direct view of angle

- Gonioprisms – Indirect gonioscope
  - Indirect view of angle
  - Light rays are reflected by the mirror
  - Prototypes – goldmann; zeiss

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Goldmann 3 mirror lens

Fig: 4

- Mirror for gonioscopy is inclined at 59° & is tongue shaped.
- Mirror to view equator is inclined at 67° and is trapezoid
- Mirror to view parsplana is inclined at 73° and is rectangular

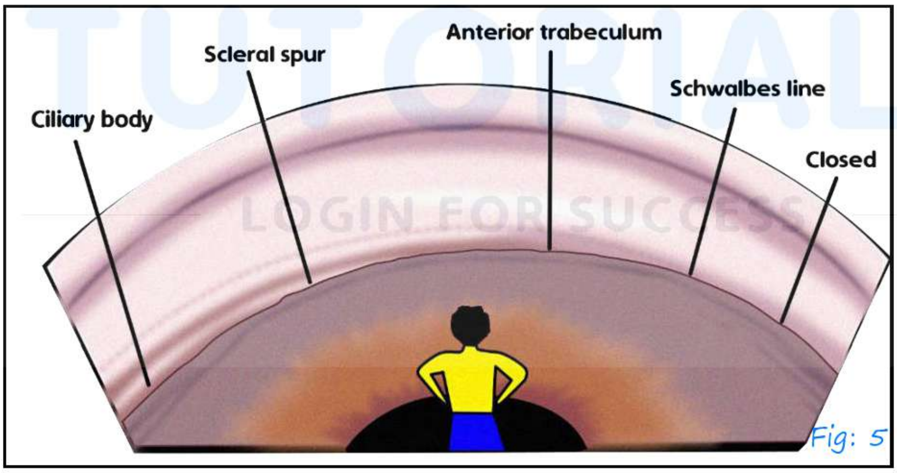
Anterior trabeculum
Scleral spur
Schwalbes line
Ciliary body
Closed

Fig: 5

- On gonioscopy → information taken is angle

  i. Open/Closed → no lines seen

  ↓

  Multiple lines seen

  ii. Abnormal accumulation/not

iii. Synechiae present/not

iv. Iris configuration

- Gonioscopy → only investigation done in small pupil
- Iridotomy
- Trabecutomy
- Arabeculectomy

→ done in small pupil

- Laser pupilloplasty

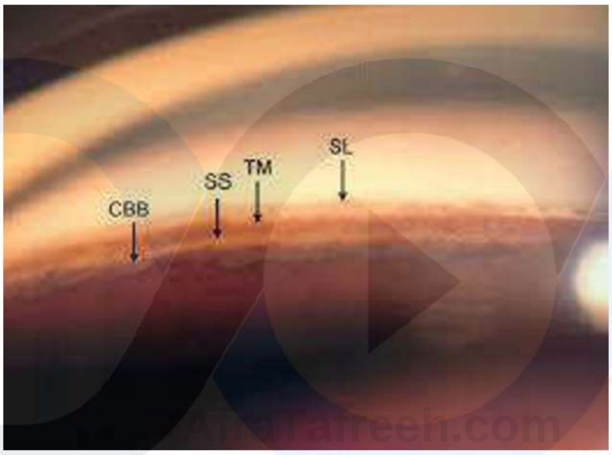

Fig: 6

- **I** can **s**till **t**ill **s**chwabe **l**ine

I – Iris

C – Ciliary body

S – Scleral spur

T – Trabecular meshwork → pigmented

S – Schwalbes line

- Schaffer's grading → Based on width of angle

ACA in degres	ACA grade	Angle status	Visible structures
0	0	Closed	No structures visible
≤ 10	1	Extremely narrow	Schwalbes line
11-19	2	Narrow	Trabecular meshwork
20-34	3	Open	Scleral spur
35-45	4	Wide open	Ciliary body

Scheies classification

- Wide open – All structure visible
- Grade I – Iris root visible
- Grade II – Ciliary body obscured
- Grade III – Posterior trabeculum obscured
- Grade IV – Only schwalbe's line visible

  - Angle depth system based on structures visualized

Speech classification

Iris Insertion	Angular approach	Peripheral iris		Pigmentation of trabecu meshwork
A Anterior to schwalbe's line	0° to 50°	R – regular	F – Flat	0 – no pigment
B Between Schwalbe's line and scleral spur		S – Steep	B – Bowed anteriorly	1+ minimal
C Scleral spur visible			P Plateau iris	2+ mild
D Deep with ciliary body visile		q queer	C – Concave	3+ moderate
E Extremely deep with >1mm of ciliary body				4+ intense

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RPC system of gradius

Grade	Structure seen
0	Closed
1	Schwalbe's line
2	Anterior (non pigmented TM)
3	Posterior (pigmented TM)
4	Scleral spur
5	Ciliary body band
6	Root of iris

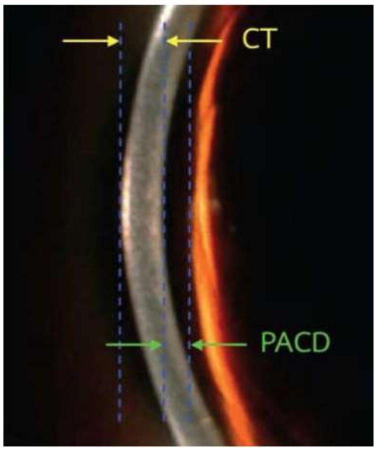

Fig: 7

Van Hericks Method 00:16:38

Van Herick's Grade	Ratio to limbal corneal section
Grade 1	<1:4
Grade 2	1:4
Grade 3	1:2
Grade 4	1:1 (or > 1:1)

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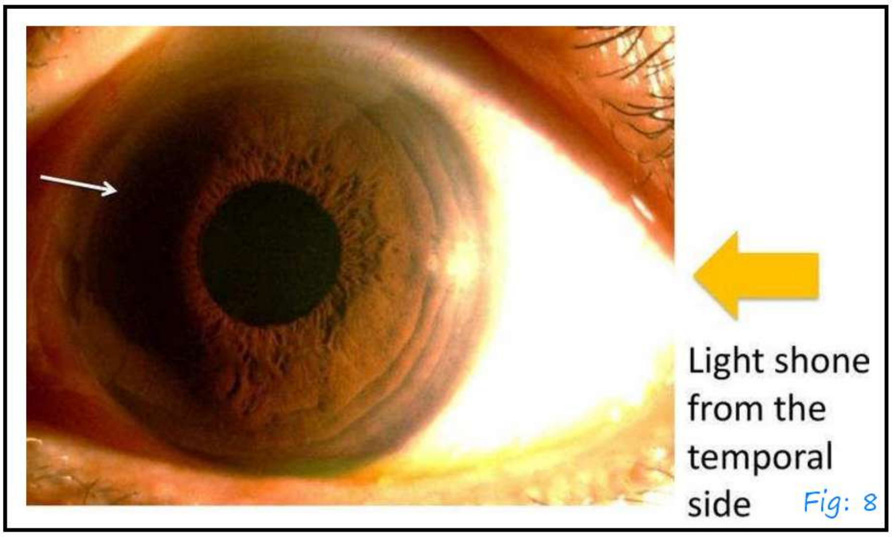
- Van Herick method → estimation of angle width

- PACD – Peripheral Anterior Chamber Depth
- Ratio of $\frac{PACD}{CT}$

Grade 4 – open angle



Light shone from the temporal side

Fig: 8

- Pen torch light is he from temporal

↓

Anterior chamber shallow

↓

Obscuration over nasal part → less illumination

↓

Eclipse sign

IOP AND TONOMETRY

- Normal value of IOP – 10-21 mmHg
- All of us have normal diurnal variation of IOP <6 mmHg
- If the diurnal variation of IOP > 6mmHg

  ↓

  Suspicion of glaucoma
- If the diurnal variation of IOP > 8 mmHg

  ↓

  Diagnostic of glaucoma
- Tonometer – instrument used to measure IOP

Types of Tonometers

i. Indentation type → causes deformation of globe by the pressure

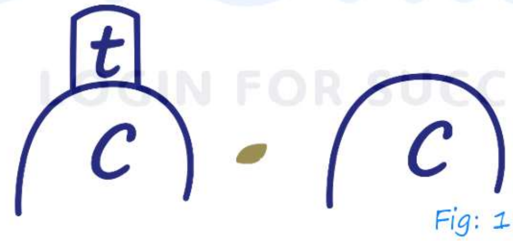


Fig: 1

Prototype → SCHIOTZ

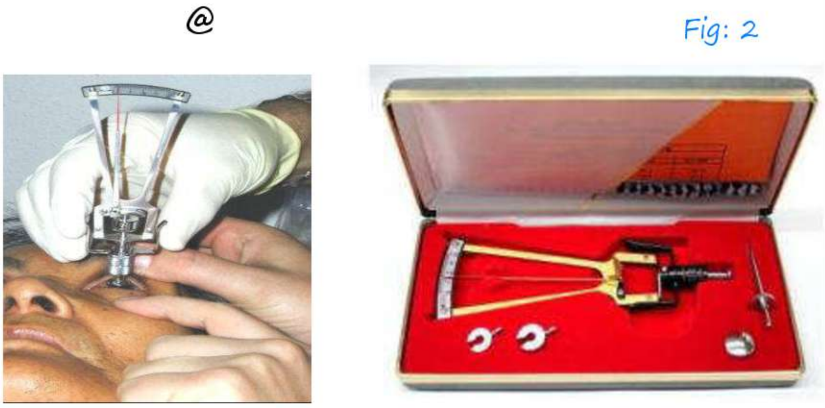


Fig: 2

Scale reading	Ocular pressure, mmHg		
	5.5g weight	7.5g weight	10.0g weight
3.0	24.4	35.8	50.6
4.0	20.6	30.4	43.4
5.0	17.3	25.8	37.2
6.0	14.6	21.9	31.8
7.0	12.2	18.5	27.2
8.0	10.2	15.6	23.1
9.0	8.5	13.1	19.6
10.0	7.1	10.9	16.5

a. After anaesthetizing the cornea, tonometer is placed over the eye → presence of 5.5m

Reading is correlated with friedenwald nomogram

Error – based on ocular rigidity

Low rigidity in high myopic

↓

False low values

b. Rebound tonometer (new tonometer)

ii. Aplantation types

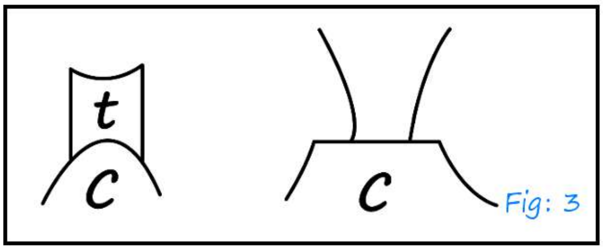


Fig: 3

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Aplantation → flattening of cornea

↓

Imbert ficks law

P = F/A

- Fixed force type → macklakov
- Fixed area type → most commonly used

↓

@ Goldman applanation tonometer

↓

Gold standard

↓

Most accurate reading

- Fixed area of 3.06mm on cornea

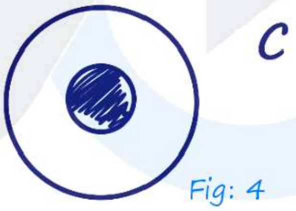


Fig: 4

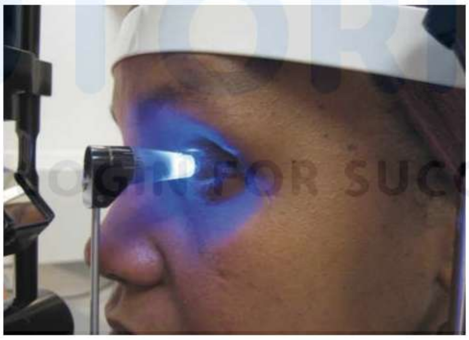


Fig: 5

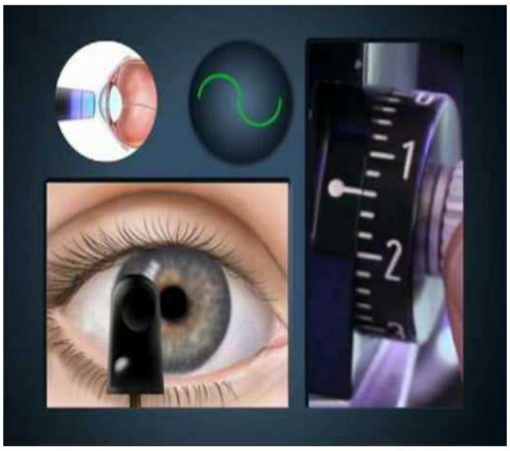


Fig: 6

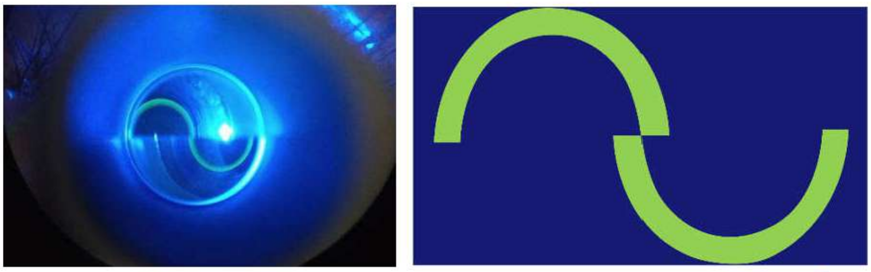


Fig: 7

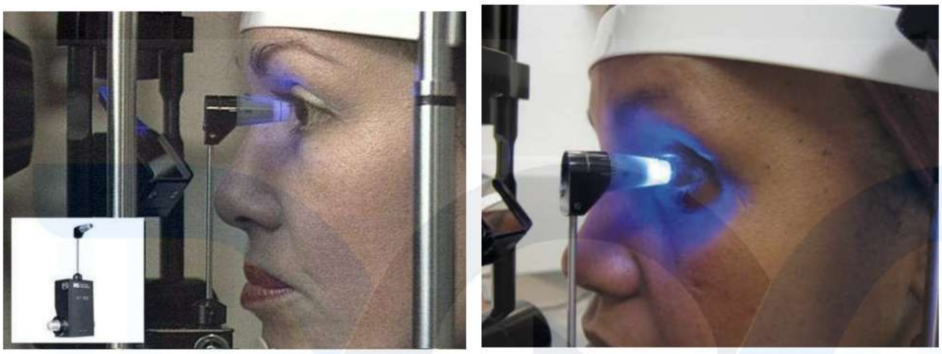


Fig: 8

- Anesthetise cornea

↓

Florescin dye

↓

Blue light from cobalt blue filter

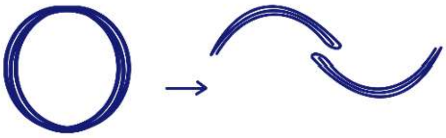
● →Green color circle

↓

Gradually turn the knob (knob mas marcicuys)

↓ 1-10 mmHg

2-20 mmHg



↓

End point → inner margins are together

IOP in the image → 14mmHg

Problem with goldman

- Patient has to sit
- Clear cornea → cornea is opaque

  ↓

  Not seen
- Cannot do screening in camps

Error with goldman

- Caliberated for a central corneal thickness of 520mm
- If cornea is 480mm & 580mm

  ↓ ↓

  False low reading   False high reading

  ↓
- Ehler's correction

  ↓

  For every 10 microns decrease in thickness, add 1 to get corrected value

Post lasik

- False low reading because corneal thickness reduced.

Falsely low IOP	Falsely High IOP
Too little fluorescein	Too much fluorescein
Thin cornea	Thick corneas
Corneal edema	Steep cornea

With the rule astigmatism 1mm Hg per 4D	Against the rule astigmatism 1m Hg per 3d
Prolonged Cataract	Wider meniscus
Repeated tonometry	Widening the lid fissure excessively
	Elevating the eyes more than 15°

Perkin Tonometer Fig: 10

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- has prism
- Hand held goldman
- Can check for children
  Bed side patients
  Screening in camps

Mackay marg tonometry Fig: 11

- Electronically generates pulses by which IOP is measured
- Uses fixed area of 6mm

TONO-PEN AVIA
Reichert
16

Tonopen Fig: 12

- Same principles → electronically generates IOP

Pneumatic tonometer Fig: 13

TUTORIALS LOGIN FOR SUCCESS

it can be used in contact lens users also

5mm silicone tube with a diaphragm gas chamber created -> electronic transducer converts air pressure into a recording scale by which IOP can be measured

Newer tonometer

↓

i. Diaton →

↓

Has prongs

diaton
I/O
diaton

Fig: 14

- Measure response if a free-falling rod as it rebounds against tarsal plate of eyelid through sclera

ii. Trigger fish

Fig: 15

↓

- Shape of contact lens has electronic chip

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iii. Pascal tonometer (pascal dynamic contour tonometry)

↓

Used in post lasik eyes because readily is not used by hystereris

↓

Ability of cornea to dissipitate energy

Fig: 16

- Pascal dynamic contour tonometry
- Contour matching instead of applantion to decrease effect of biomechanics
- Piezoelectric crystals

iv. ORA ocular response analyser

Fig: 17

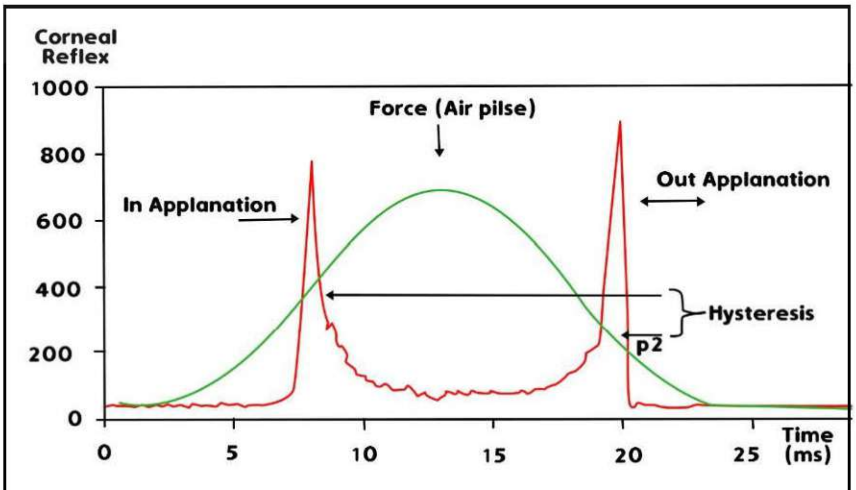


Fig: 18

  - Based on applanation
  - Applanation is done twice – one when touches cornea

Rebound tonometer

- Indention type of tonometer

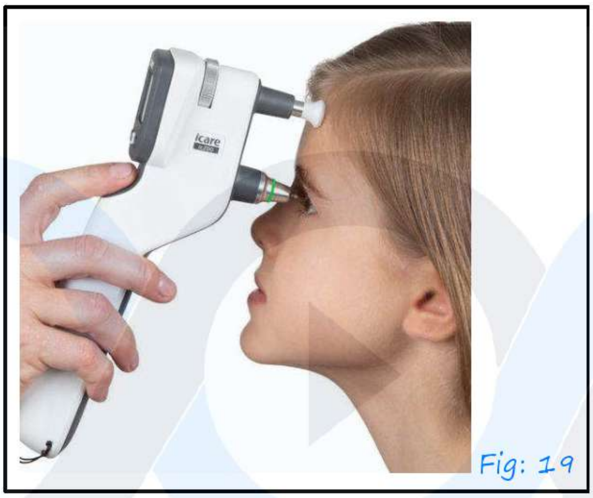


Fig: 19

- I CARE tonometer
- Electromagnetic field
- 1.8mm plastic ball on stainless wire placed under EM field.
- It hits the cornea and decelerate
- If more – high IOP
- Affected by – biomechanical properties of cornea.
- Speed of deceleration is measured
- Can be used as home/self-tonometer
- Upper one placed on scalp
- Lower one going to indent cornea

Optic Disc Examination 00:28:30

Fig: 20

Optic disc can be examined by

i. Opthalmoscope
    - Direct
    - Indirect

ii. Slit lamp biomicroscopy (best)

↓

90D> 60 D> 30D lens used

↓

Best

iii.

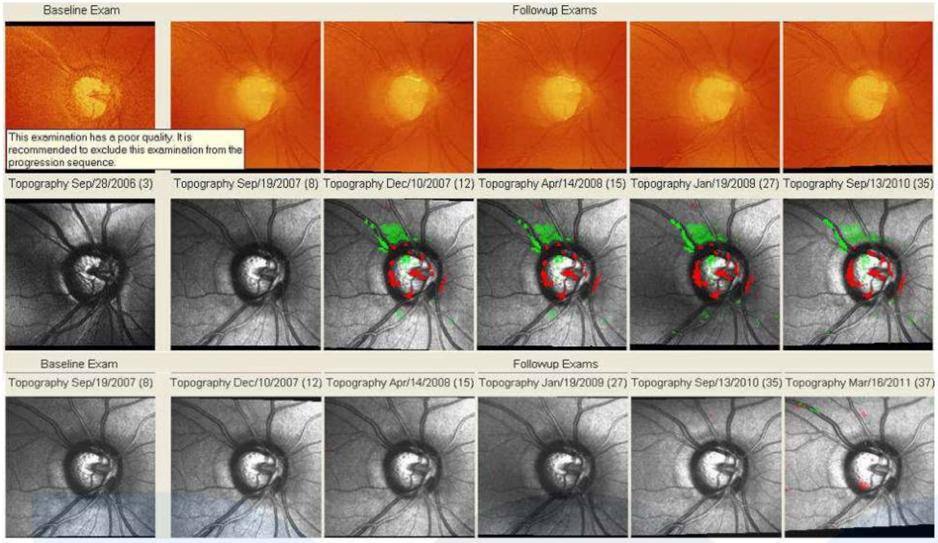
Fig: 21

→ Hruby lens → -58D

iv. Confocal scanning laser tomography

CSLT

00:30:04



This examination has a poor quality. It is recommended to exclude this examination from the progression sequence.

Baseline Exam
Followup Exams
Topography Sep/28/2006 (3)
Topography Sep/19/2007 (8)
Topography Dec/10/2007 (12)
Topography Apr/14/2008 (15)
Topography Jan/19/2009 (27)
Topography Sep/13/2010 (35)
Baseline Exam
Followup Exams
Topography Sep/19/2007 (8)
Topography Dec/10/2007 (12)
Topography Apr/14/2008 (15)
Topography Jan/19/2009 (27)
Topography Sep/13/2010 (35)
Topography Mar/16/2011 (37)

Fig: 22

↓

- Based on Heidelberg tomography
- View of optic disc and para papillary retinal nerve fibre layer.

GLAUCOMA TYPES MECHANISM & CLASSIFICATION

- But glaucoma is a group of disorders which cause chronic progressive optic neuropathy with accompanying visual field defects
- Increases IOP → most important risk factor
- Increases IOP + optic nerve defect + visual field change

High IOP

↑

i. Aqueous is increasingly secreted (rase)

Epidemic dropsy

ii. Decreased drainage → open angle mechanism

→ closed angle mechanism

→ congenital glaucoma

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Theories related to glaucoma 00:03:16

i. Mechanical – structural damage to angle structures & lamina cribrosa

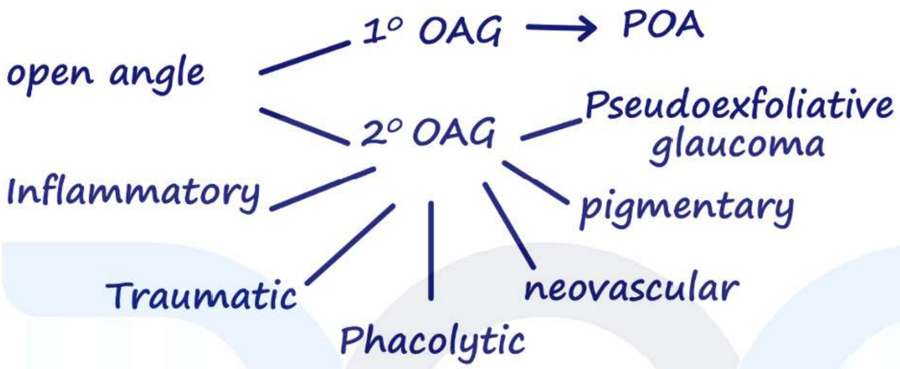
ii. Vascular → decreased perfusion around optic nerve head

↓

Lamina cribrosa is not structurally strengthened

Simplified classification of glaucoma 00:05:06

- Open angle
  - 1° OAG → POA
  - 2° OAG


open angle
1° OAG
POA
2° OAG
Pseudoexfoliative glaucoma
Inflammatory
pigmentary
Traumatic
neovascular
Phacolytic

Closed angle

1° AC ↓
Anatomically predisposed

2° AC ↓
- Malignant
- Phacomorphic
- Neovascular

- Congenital glaucoma

OPEN ANGLE GLAUCOMA

- Angle is open on gonioscopy → normal

Mechanism

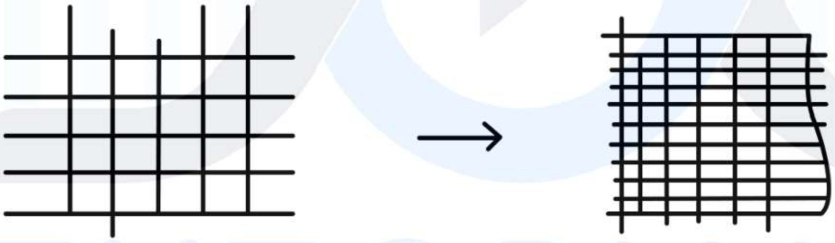
- As we grow old there is shrinkage of trabecular meshwork → Gradual decrease in drainage of aqueous

↓

Chronic rise of IOP

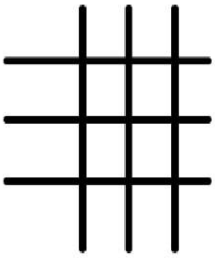
↓

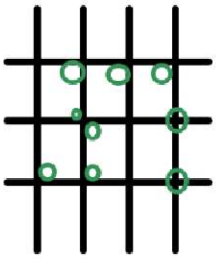
POAG/Chronic simple glaucoma



2° open angle glaucoma

- HMSC/ phacolytic/ neovascular/uvetic
- Trabecular mesh work normal
- These accumulation of cells like phacolytic, new vessels, inflammatory cells





Mechanism 00:04:28

- Age related sclerosis of pores of TM
- Deposition of ECM in TM
- Structural changes
  - Loss of phacolytic activity
  - Decrease in pore size of schlemm's canal

Risk factors 00:04:52

- POAG → Also known as chronic simple glaucoma
- According to ocular hypertension treatment study
  - Advancing age
  - Male
  - Black
  - Myopia
  - Vertical CD ratio
  - Thin cornea
  - Family history
  - Myocilin gene
- Only modifiable risk factor – High IOP

Clinical features 00:07:40

- Mostly asymptomatic
- 1.Gradual painless progressive decrease in vision
- 2.Repeated change of presbyopia correction
- 3.Increased dark adaptation time

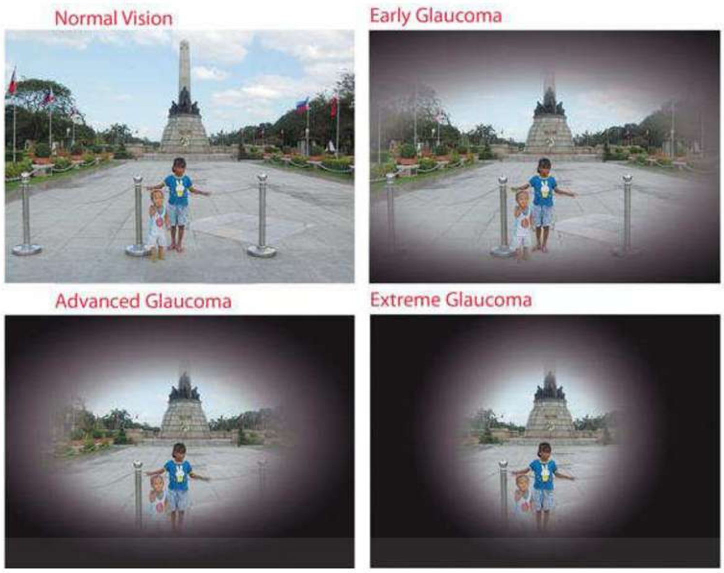
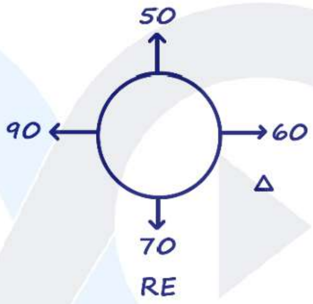

Normal Vision
Early Glaucoma
Advanced Glaucoma
Extreme Glaucoma

Fig: 1


50
90
60
70
△
RE

- In glaucoma constriction of field → Tumor vision

Signs 00:11:25

- ↑ IOP
- Optic nerve changes

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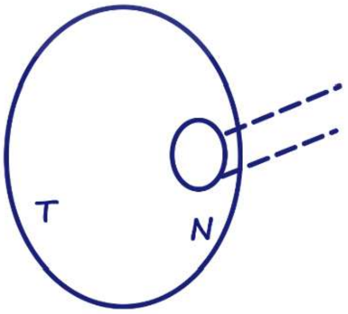
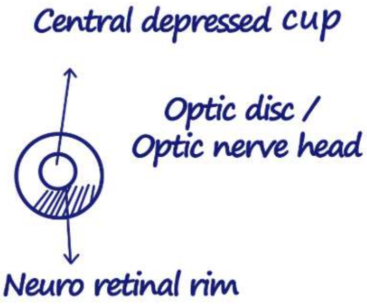
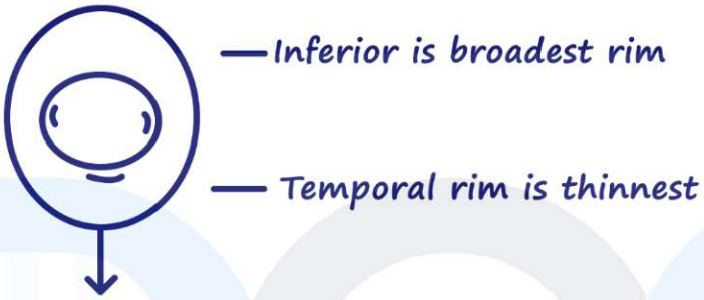
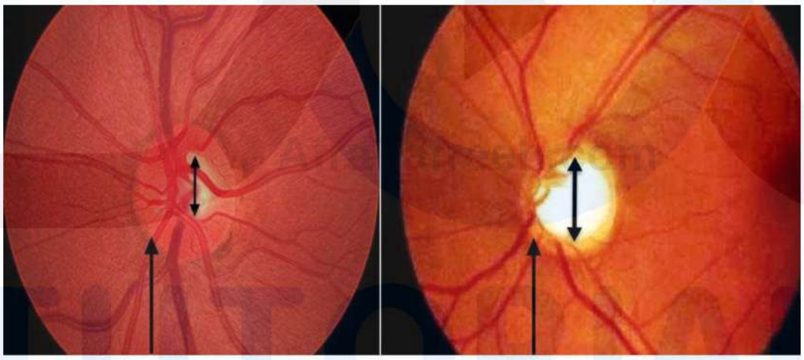



Central depressed cup
Optic disc /
Optic nerve head
Neuro retinal rim

- Optic nerve is made of ganglion cells
- Central part does not have ganglion cells

- (N) cup to disc ratio 0.3 or 0.4 up to 0.6:1 is also considered normal
- Cup to disc ration – Glaucoma & Also seen in myopes
- 0.7:1 → (N) seen in 15% of (N) individuals
- In cup disc ration – ISNT rule is followed


Inferior is broadest rim
Temporal rim is thinnest



Normal optic nerve head

Glaucomatous cupping

Fig: 2

Changes in optic nerve head

- In glaucoma ganglion cells are lost → Neuro retinal rim becomes thin
- Because of mechanical pressure cup is also deepened
- Vertical cup ratio increased

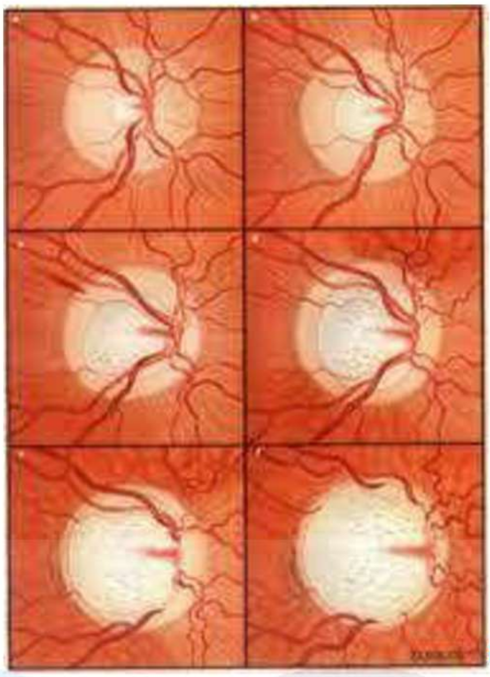


Fig: 3

DOC TUTORIALS LOGIN FOR SUCCESS

- Vertically cup oval
- Large cup disc ratio > 0.7:1
- Disc hemorrhage H1N, LAB 3
- Notching
- Laminar dot sign
- Asymmetry
- Bayonetting sign
- Baring of circumlinear vessels
- Bean pot cupping

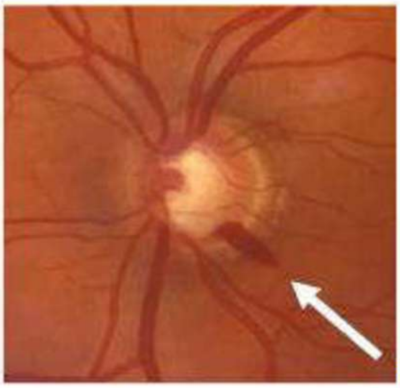


Fig: 4

- Disc hemorrhage
- Drance hemorrhage
- Flame shaped Hemorrhage at the disc
- Most commonly infer temporal
- More common in normal tension glaucoma

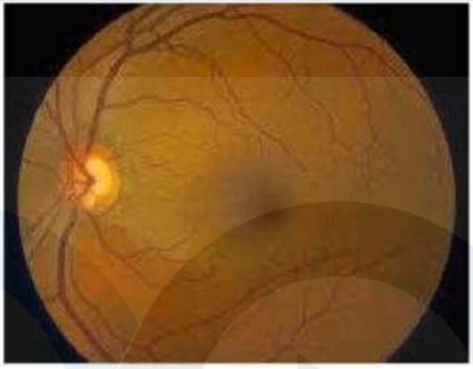
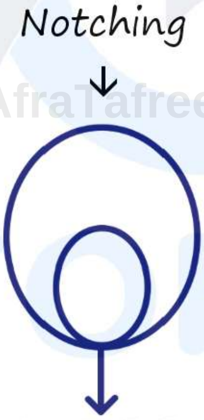


Fig: 5



Complete absence of NRR from plane → notching

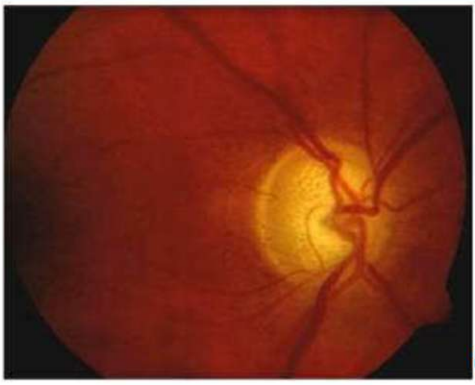


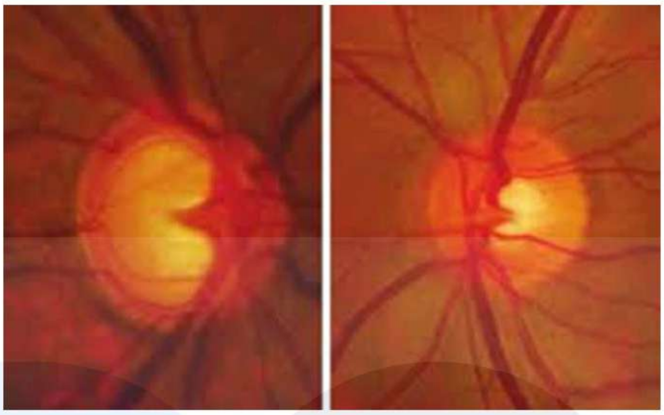
Fig: 6

DOC TUTORIALS LOGIN FOR SUCCESS

Laminar dot sign

↓

Visibility of pores of lamina cribrosa



Asymmetry of CD ratio >0.2 Fig: 7

Baring

- Circumlinear vessels are small branches arising from the retinal vessels.
- Only visible in 50% of normal eyes.
- These vessels follow the curvilinear path and run along the superior and inferior margins of the optic cup.
- Baring occurs with enlargement of the of the cup and pallor.
- The rim narrows and the loss of tissue leaves these vessels isolated and “bared.”

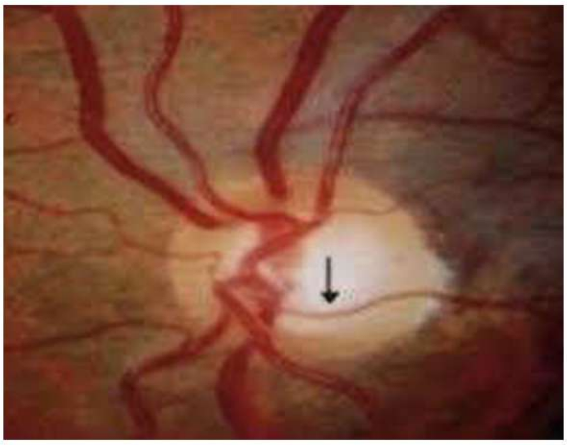


Fig: 8

BAYONETTING

- With advancement of cupping, the vessels emerge from the floor of the cup and then ascend up the steep wall of the cup under the overhanging edge.
- The vessels are not visible under the overhanging edge.
- The vessels emerge again at the disc margin and make a sharp bend that can resemble a bayonet

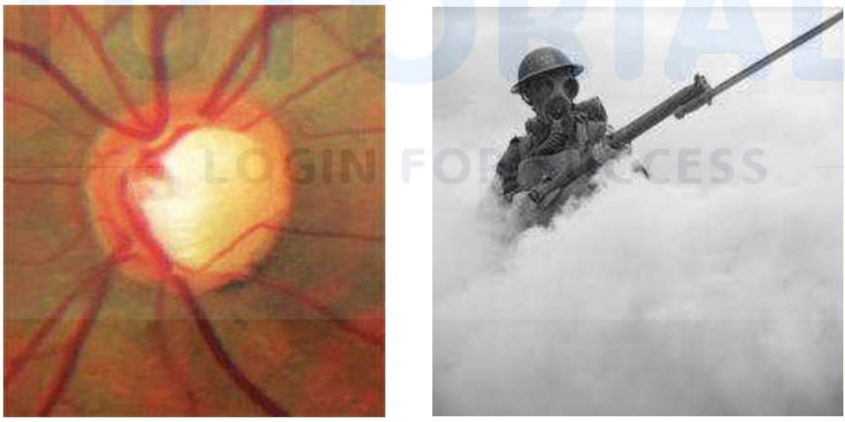
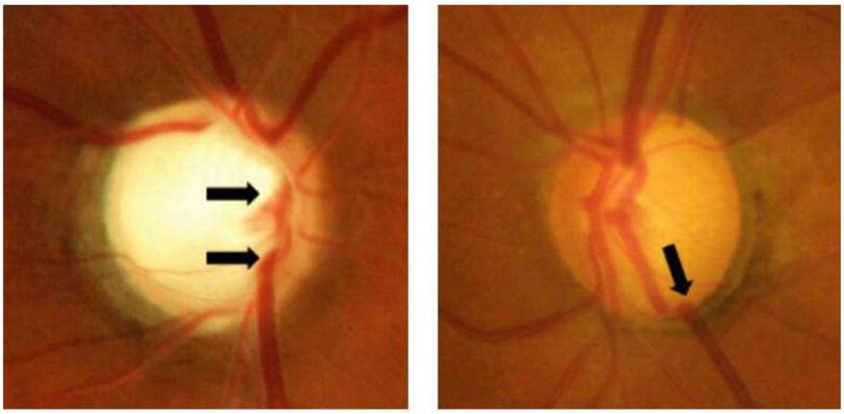


Fig: 9

TUTORIALS
AnaTaleem.com
LOGIN FOR ACCESS



Nasal shift bayonetting Fig: 10

Bean pot cupping

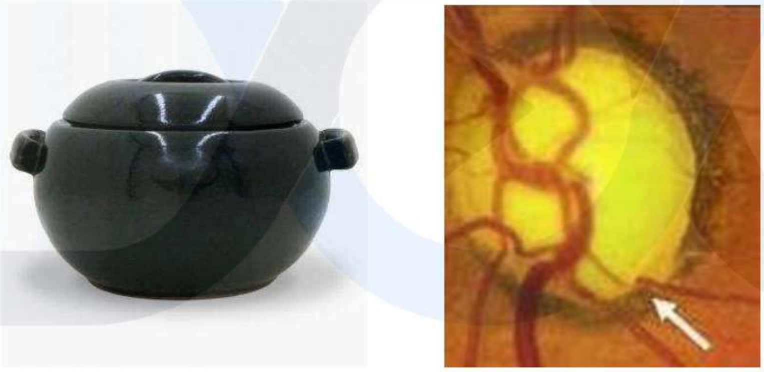


Fig: 11

TUTORIALS LOGIN FOR SUCCESS

Alpha zone

- Hypo and hyper pigmented areas
- Present in normal as well as in glaucomatous eyes

Beta zone

- Atrophy of the retinal pigment epithelium and choriocapillaris
- Large choroidal vessels become visible
- More common in glaucomatous eyes

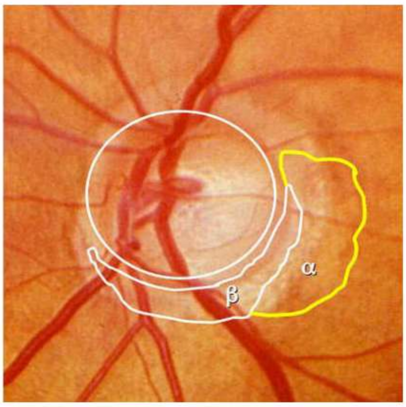


Fig: 12

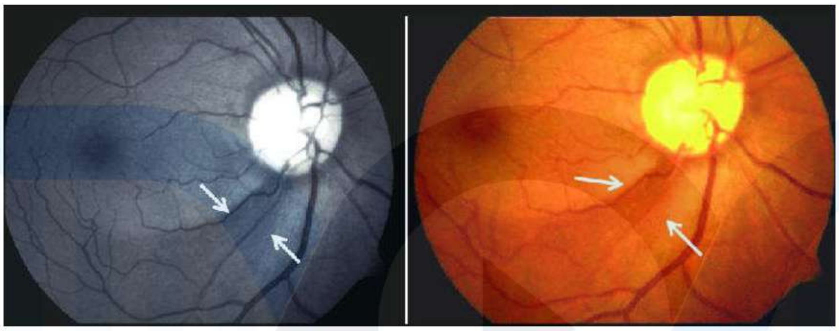


Fig: 13

RNFL defects

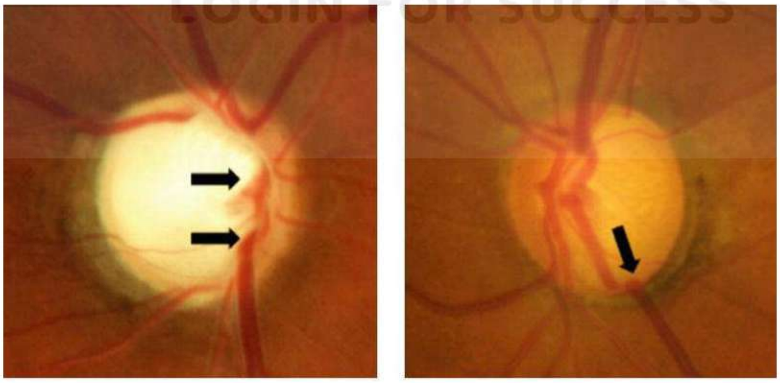
↓

Retinal nerve fibres layer defects

↓

wedge shaped

AfraTafreen.com
TUTORIALS
LOGIN FOR SUCCESS



Nasal shift

bayonetting

Fig: 14

Specific Signs

1. Vertically oval cup > Horizontally oval cup
2. Bayonetting sign
3. RNFL Wedge shaped defects

VISUAL FIELD ASSESSMENT

Clinical features 00:00:16

- IOP
- Optic nerve changes
- Visual field changes

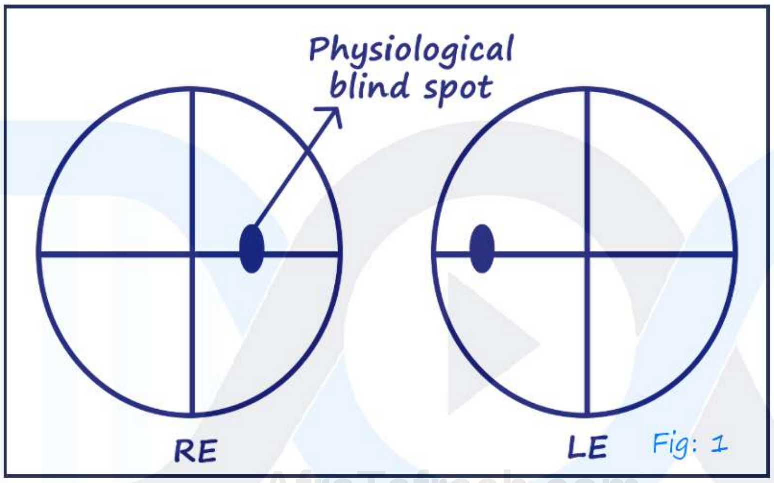


Fig: 1

DOC TUTORIALS LOGIN FOR SUCCESS

- Absolute -ve scotoma – area in visual field which is approximately 10-15 degress from centre
- Blind spot – Corresponds to optic disc → Optic disc does not have photoreceptors
- Present on temporal side

Changes in glaucoma 00:04:03

- Perimeter – used to find visual field/ change

Types

Static ↓ Non moving target ↓
Humphreys
Octopus

Kinetic ↓ Moving target ↓
1.Goldman
2. Lister
3. Tangenic screen

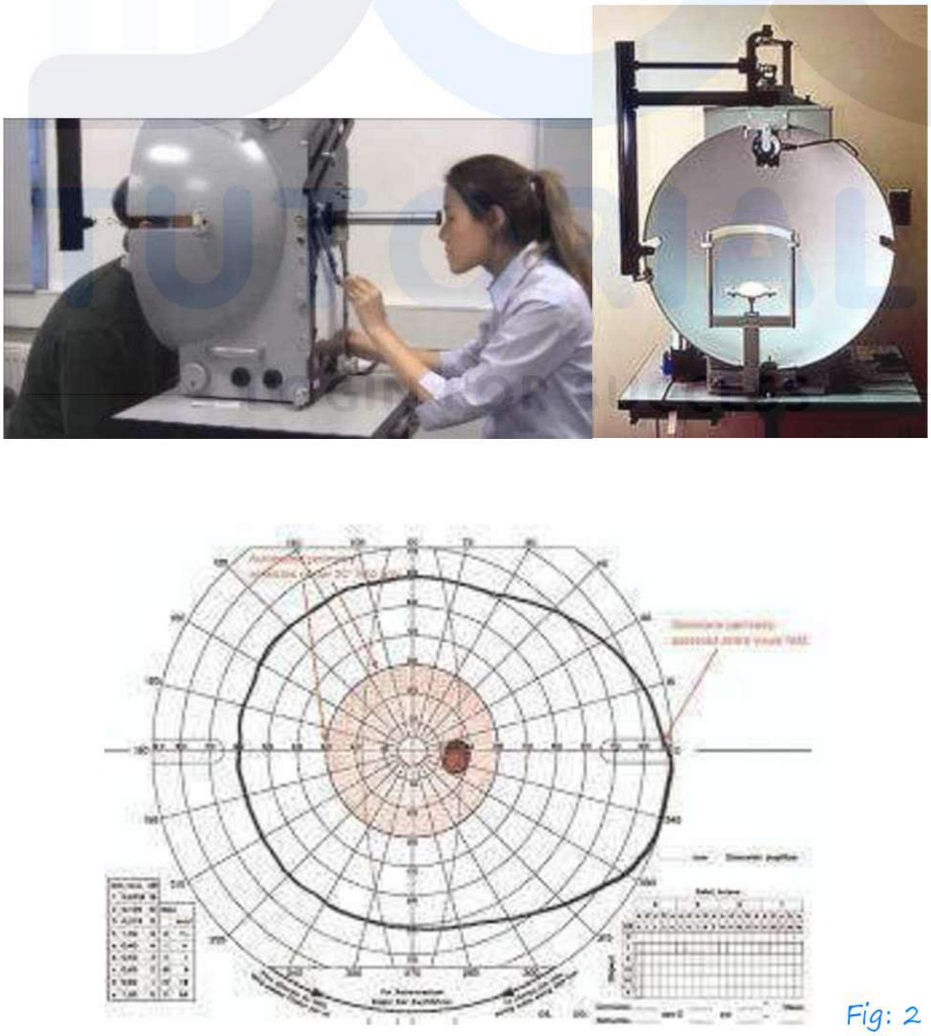


Fig: 2

Lister perimeter

Fig: 3

Perimetry: Humphrey's

Fig: 4

TUTORIALS LOGIN FOR SUCCESS

- White – White perimetry
- Patient is asked to look at fixation target
- After few second stimulus is given
- Patient should pick the stimulus
- If the stimulus is not picked up when you print out

  → Black area would be there → Scotoma
- Target in size 3

- Start with 0.25mm → 1mm → 4mm
- Strategy → SITA → Swedish interactive threshold algorithm
- Less time consuming

Arrangement of Fibres in Retina 00:10:10

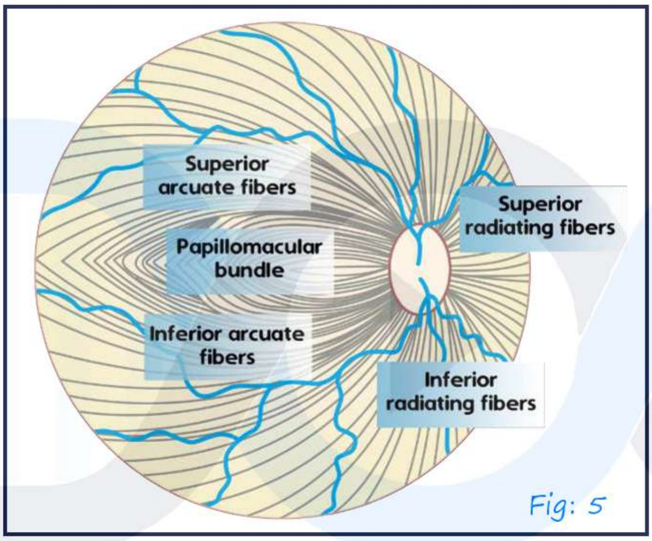
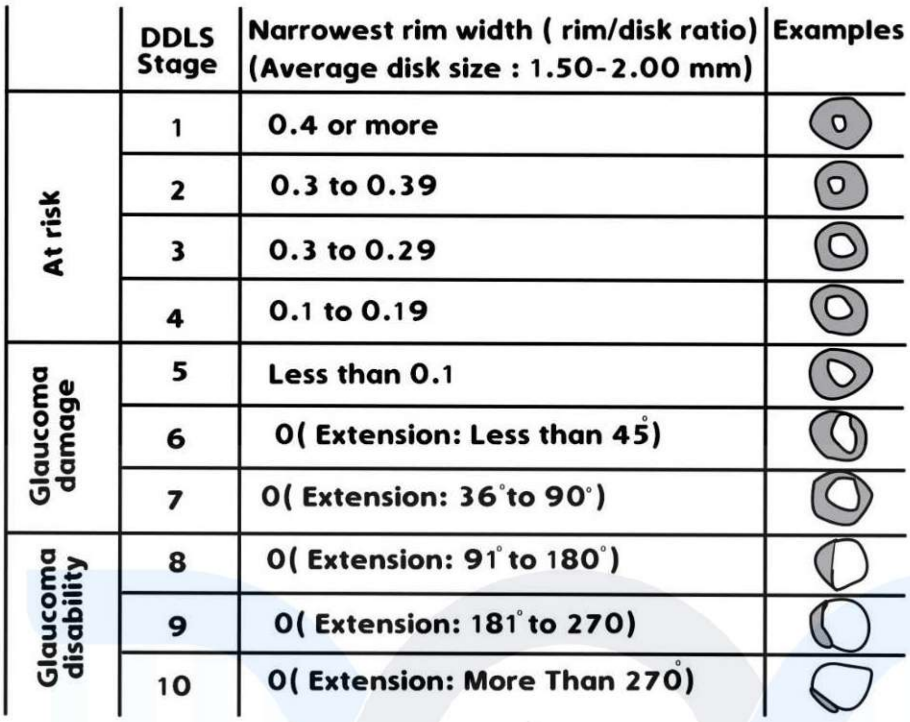

Superior arcuate fibers
Papillomacular bundle
Inferior arcuate fibers
Superior radiating fibers
Inferior radiating fibers

Fig: 5

- Nasal fibres of retina comes as superior and inferior radiating fibres
- Fibres from temporal region hit the disc superiorly and inferiorly → Superior arcuate fibres & Inferior arcuate fibres
- Earlier to be damaged in glaucoma → Arcuate fibres
- Fibres from macula and conversing on temporal side of disc → papillomacular bundle
  - Most resistant in glaucoma
  - First to go when we have ischemia/trauma → Central scotoma



	DDLS Stage	Narrowest rim width (rim/disk ratio) (Average disk size : 1.50-2.00 mm)	Examples
At risk	1	0.4 or more	
	2	0.3 to 0.39	
	3	0.3 to 0.29	
	4	0.1 to 0.19	
Glaucoma damage	5	Less than 0.1	
	6	0(Extension: Less than 45°)	
	7	0(Extension: 36°to 90°)	
Glaucoma disability	8	0(Extension: 91° to 180°)	
	9	0(Extension: 181° to 270)	
	10	0(Extension: More Than 270°)	

- DDLS – Disc damage likelihood scale

Visual field changes 00:16:00

- Isopteric contraction

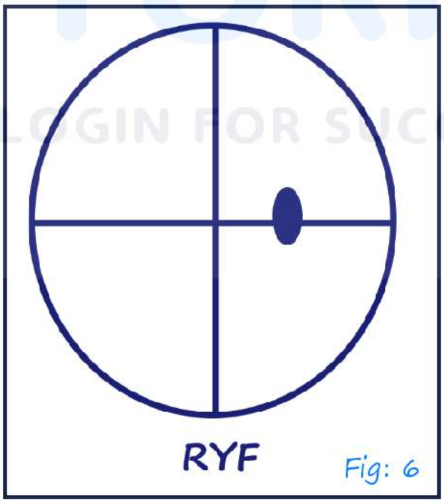
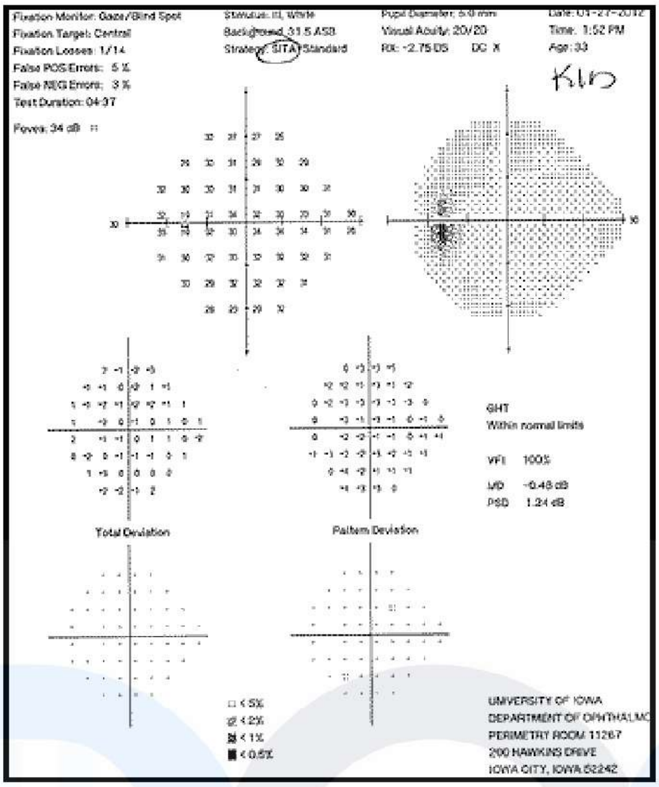


Fig: 6



Fixation Monitor: Gaze/Blind Spot
Fixation Target: Central
Fixation Losses: 1/14
False POS Errors: 5 %
False NEG Errors: 3 %
Test Duration: 04:37

Fovea: 34 dB

Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA Standard

Pupil Diameter: [illegible]
Visual Acuity: 20/20
RX: -2.75 DS DC X

Date: [illegible]
Time: 1:52 PM
Age: 33

Klo

GHT
Within normal limits

VFI 100%

MD -0.48 dB
PSD 1.24 dB

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DEPARTMENT OF OPHTHALMO
PERIMETRY ROOM 11267
200 HAWKINS DRIVE
IOWA CITY, IOWA 52242

- Generally, perimetry done at 24-2/30-2
- Slight contraction of visual field
- Not a clinically significant/identifiable sign

- Baring of blind spot
  - Some constriction of visual field
  - Blind spot is out of boundary
  - Exclusion of blind spot from field
- Paracentral wing shaped scotoma
  - Generally, occurs in supero nasal quadrant because inferotemporal fibres are first to be affected
  - 1st visually significant scotoma
  - All scotoma is seen in bjerrums area → 10-25° from centre
- Seidel seotoma – Sickle shaped /Derringer scotoma

- Extension of blind spot joining the paracentral scotoma in sickle shape manner

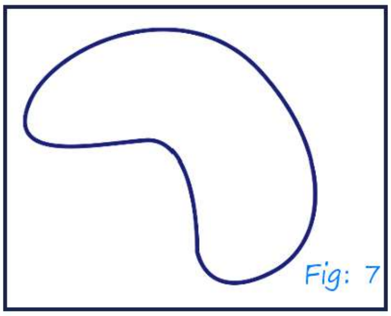


Fig: 7

- Arcuate scotoma

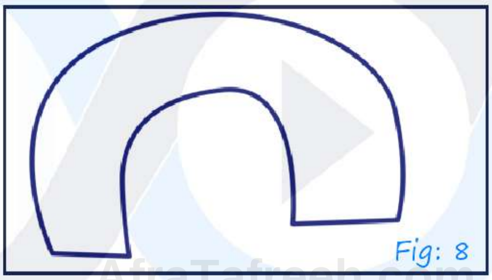


Fig: 8

- Double arcuate scotoma/ Dough nut scotoma
  - aka Tunnel vision ring scotoma
  - D/D → Retinitis pigmentosa

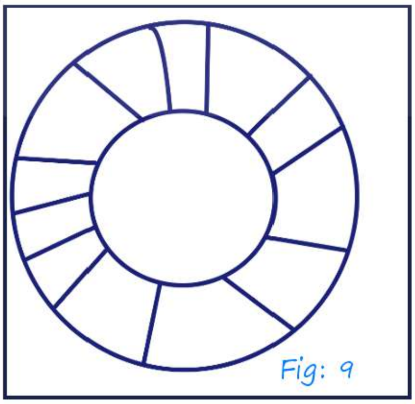


Fig: 9

- Roenne nasal step
  - Because unequal damage of fibres

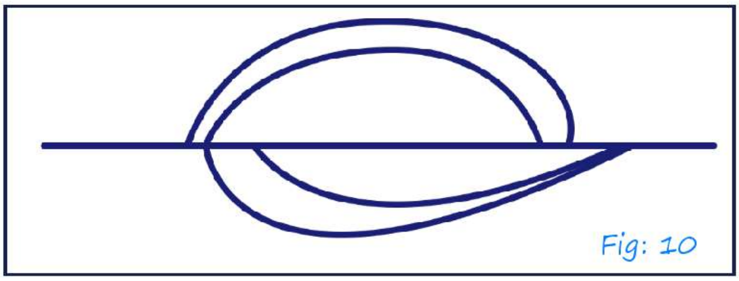


Fig: 10

- Differential loss of fibres
- Central vision is preserved → Tunnel vision
- Sometimes central vision is also lost
- Small temporal island of vision is left

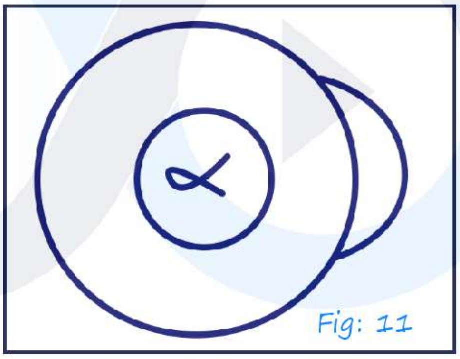
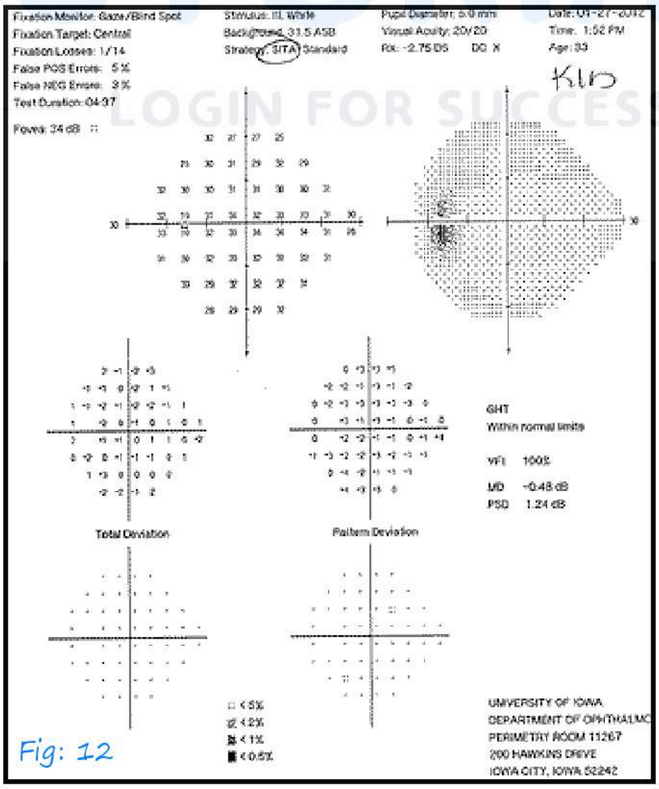


Fig: 11

DOC TUTORIALS
LOGIN FOR SUCCESS



Fixation Monitor: Gaze/Blind Spot
Fixation Target: Central
Fixation Losses: 1/14
False POS Errors: 5 %
False NEG Errors: 3 %
Test Duration: 04:37

Fovea: 34 dB

Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA Standard

Pupil Diameter: 5.0 mm
Visual Acuity: 20/20
Rx: -2.75 DS DC X

Date: 01-27-2012
Time: 1:52 PM
Age: 33

Klb

GHT
Within normal limits

VFI 100%

MD -0.48 dB
PSD 1.24 dB

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200 HAWKINS DRIVE
IOWA CITY, IOWA 52242

Fig: 12

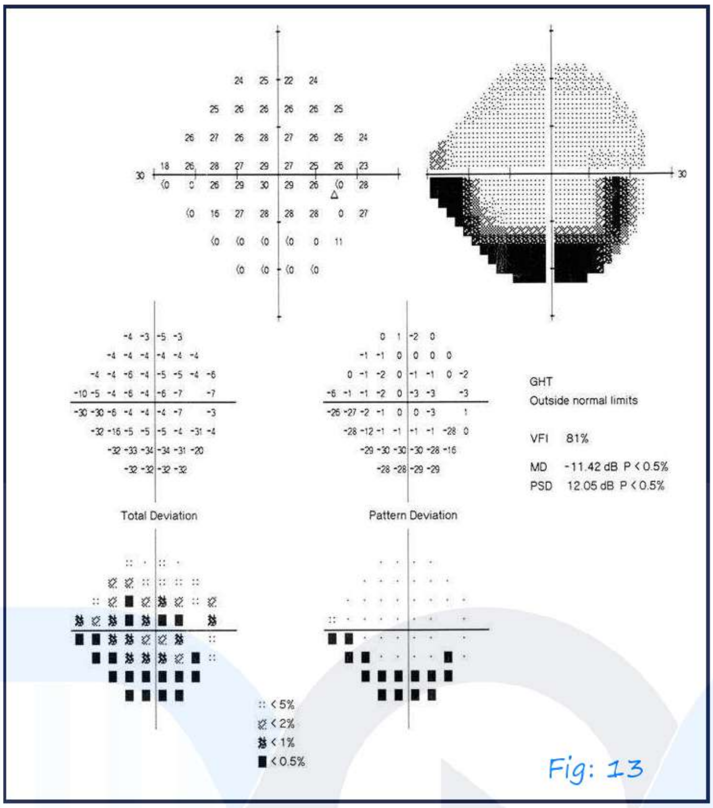

24 25 22 24
25 26 26 26 26 25
26 27 26 28 27 26 26 24
18 26 28 27 29 27 25 26 23
30
<0 0 26 29 30 29 26 <0 28
<0 16 27 28 28 28 0 27
<0 <0 <0 <0 0 11
<0 <0 <0 <0
30
-4 -3 -5 -3
-4 -4 -4 -4 -4 -4
-4 -4 -6 -4 -5 -5 -4 -6
-10 -5 -4 -6 -4 -6 -7 -7
-30 -30 -6 -4 -4 -4 -7 -3
-32 -16 -5 -5 -5 -4 -31 -4
-32 -33 -34 -34 -31 -20
-32 -32 -32 -32
Total Deviation
0 1 -2 0
-1 -1 0 0 0 0
0 -1 -2 0 -1 -1 0 -2
-6 -1 -1 -2 0 -3 -3 -3
-26 -27 -2 -1 0 0 -3 1
-28 -12 -1 -1 -1 -1 -28 0
-29 -30 -30 -30 -28 -16
-28 -28 -29 -29
Pattern Deviation
GHT
Outside normal limits
VFI 81%
MD -11.42 dB P < 0.5%
PSD 12.05 dB P < 0.5%
:: < 5%
▨ < 2%
▩ < 1%
■ < 0.5%

Fig: 13

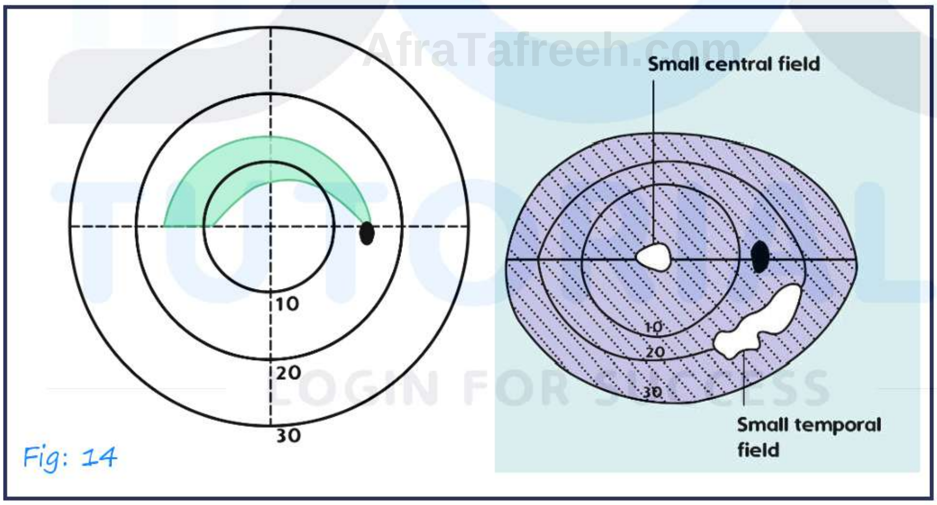

10
20
30
Small central field
10
20
30
Small temporal field

Fig: 14

Captimetry 00:28:05

- Type of perimetry which targets central 5 degree of visual field
- 80% of the ganglion cells are parvocellular P type cells sensitive to color and contrast

- 5% are koniocellular K cells, responsive to blue – yellow opponents
- 15% are magnocellular M cells that react to temporally modulated stimuli

Pre – Penmetric analysis 00:31:04

Blue on yellow perimetry

- Short Wavelength Automated Perimetry (SWAP)
- These small ganglion cells connect to the koniocellular pathway in the lateral geniculate ganglion.
- only 5-10% of all retinal ganglion cells.
- yellow background light suppresses red and green cones while blue cones are stimulated by a blue stimulus

TUTORIALS LOGIN FOR SUCCESS

Fig: 15

Frequency doubling perimeter

- Based on a flicker illusion created by flickering of a low spatial frequency sinusoidal grating at a high temporal frequency.

- *Creates an image that appears double its actual spatial frequency.*
- *Early glaucomatous nerve damage is thought to be due to loss of large-diameter*
- *RGC the M cells of the magnocellular layer.*

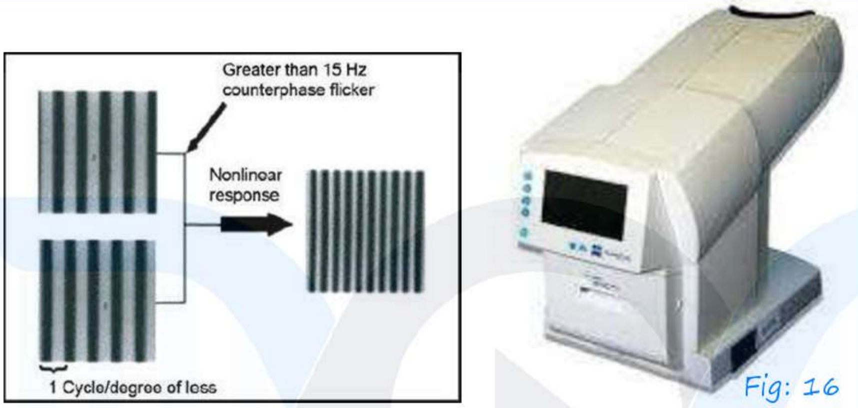
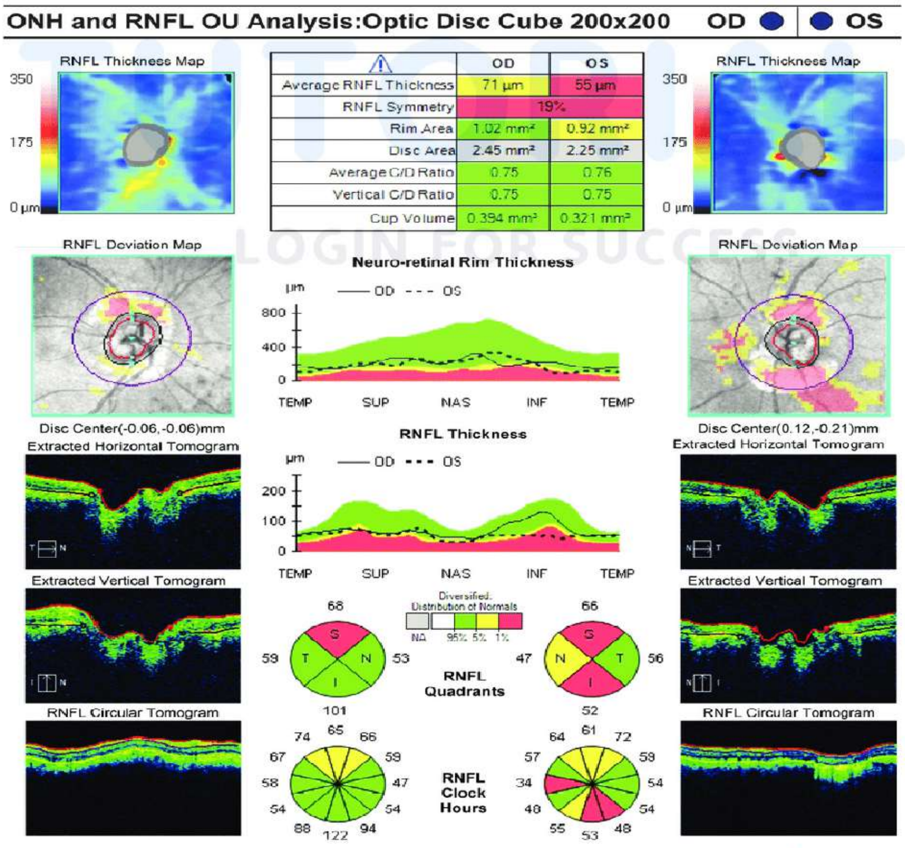

Greater than 15 Hz counterphase flicker
Nonlinear response
1 Cycle/degree of loss

Fig: 16

RFNL thickness analysis 00:33:50



ONH and RNFL OU Analysis:Optic Disc Cube 200x200 OD ● | ● OS

⚠	OD	OS
Average RNFL Thickness	71 µm	55 µm
RNFL Symmetry	19%	
Rim Area	1.02 mm²	0.92 mm²
Disc Area	2.45 mm²	2.25 mm²
Average C/D Ratio	0.75	0.76
Vertical C/D Ratio	0.75	0.75
Cup Volume	0.394 mm³	0.321 mm³

RNFL Thickness Map
350
175
0 µm
RNFL Deviation Map
Neuro-retinal Rim Thickness
µm
OD
OS
800
400
0
TEMP
SUP
NAS
INF
TEMP
Disc Center(-0.06,-0.06)mm
Extracted Horizontal Tomogram
Disc Center(0.12,-0.21)mm
RNFL Thickness
200
100
Extracted Vertical Tomogram
RNFL Circular Tomogram
Diversified: Distribution of Normals
NA
95%
5%
1%
RNFL Quadrants
68
59
53
101
66
47
56
52
S
T
N
I
RNFL Clock Hours
65 66 59 47 54 94 122 88 54 58 67 74
61 72 59 54 54 48 53 55 48 34 57 64

Fig: 17

VARIANTS IN GLAUCOMA & NORMAL TENSION GLAUCOMA

Variants in glaucoma

i. Occular HTN → high pressure (increases IOP) + optic nerve normal+ visual field also normal
   Eg. IOP – 25 mmHg
ii. Normal tension glaucoma
   IOP is normal. If IOP is normal+ optic nerve damaged+ visual field damaged.
   Eg. IOP – 18 mmHg

- Pathophysiology – Lamina cribrosa is not strengthened
  ↓
  Cannot handle normal pressure
  ↓
  Loss of neurotrophins
  - Perfusion @ optic nerve head is not appropriate

Risk factors

i. Migraine
ii. Nocturnal hypotension
iii. Raynauds disease

- Collaborative normal tension glaucoma
  ↓
  If SOP is decreased from 30% of baseline to glaucoma decreased

Clinical features

i. Drance hemorrhages (most common)

ii. Visual field defects are more towards centre

Treatment

Prostaglandin analogues → DOC
↓
BIMATOPROST
↓
Neuroprotective action

- Brimomidine ⎤ Neuroprotective action
- Dorzolamide ⎦
- Rho kinase inhibitor
- Calcium channel blockers
- Oral memantin

ANGLE CLOSURE GLAUCOMA

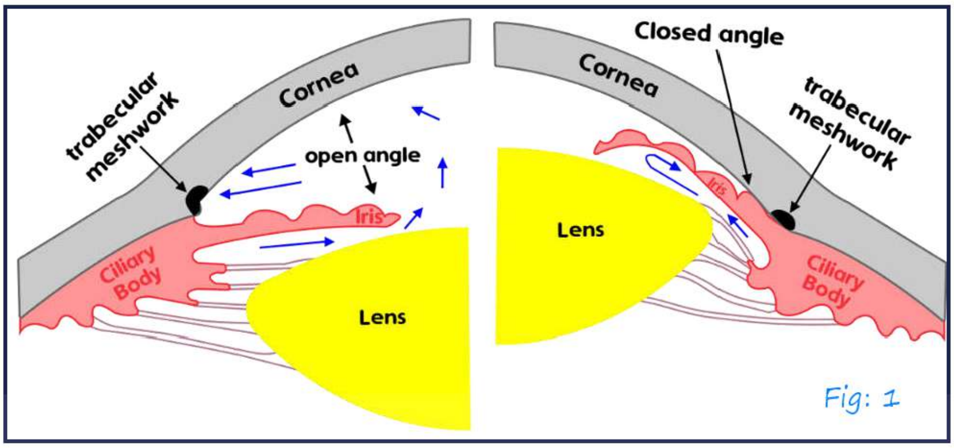

trabecular meshwork
Cornea
open angle
Iris
Ciliary Body
Lens
Closed angle
Cornea
trabecular meshwork
Iris
Lens
Ciliary Body

Fig: 1

- Angle closure glaucoma occurs in anatomically predisposed individuals
- Axial length <24mm
- Smaller cornea
- Hypermetropia
- Iris lens diaphragm forward shift
- Normal or large lens

More common in females than males

Precipitations factors 00:01:52

- Mid dilated pupillary position
  - Cycloplegic , mydriatic drugs
  - Stress
  - Dark room
  - Emotional
  - Near work

TUTORIALS LOGIN FOR SUCCESS

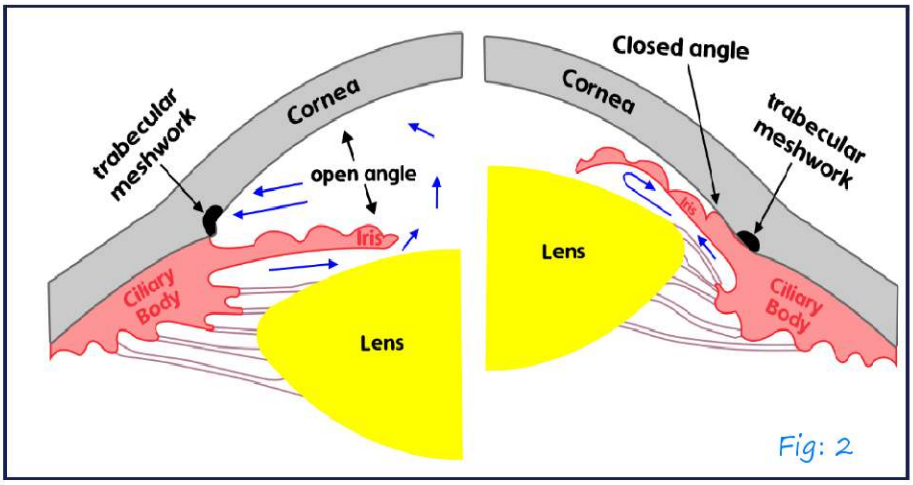

trabecular meshwork
Cornea
open angle
Iris
Ciliary Body
Lens
Closed angle
Cornea
trabecular meshwork
Iris
Lens
Ciliary Body

Fig: 2

- Crowding of anterior segment
- Anterior chamber shallow
- Mid dilated pupil → Iris and lens will touch each other

↓

Relative pupillary block

↓

Collection of aqueous in posterior chamber

↓

Iris pushed to peripheral part

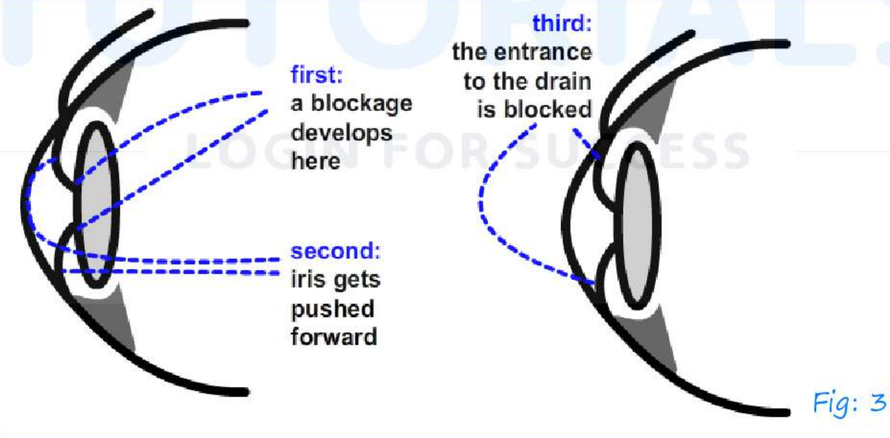

first:
a blockage develops here
second:
iris gets pushed forward
third:
the entrance to the drain is blocked

Fig: 3

Mechanism 00:04:56

Mid dilated pupil
↓
Increase apposition between lens and iris
↓
Relative pupillary block → Aqueous collects in PC

Appositional angle closure → Long standing
↓
Pushes iris peripherally towards angle
↓
Aqueous collects in PC

Long standing
↓
Synaechial angle closure

Stage	Definition
Primary angle closure suspect (PACS)	Iridotrabecular contact 180° but no evidence of trabecular mesh work or optic nerve damage
Primary angle closure (PAC)	Iridotrabecular contract 180° with elevated IOP or PAS but no optic nerve damage
Primary angle closure glaucoma (PACG)	PAC with glaucomatous optic neuropathy

- In primary angle closure suspect primary angle closure
  → Prophylactic peripheral iridotomy

Symptoms 00:09:41

- Acute congestive glaucoma/Angle closure

Clinical features

- Pain
- Redness in eye
- Decrease in vision
- Headache
- Jaw pain
- Nausea /vomiting
- IOP ↑

Signs 00:10:56

- Cornea - hazy
- Epithelial and stromal edema
- Decreased vision
- Redness (+)
- Pupil → vertically oval mid dilated non reacting to light and accommodation
- IOP ↑↑ – By digital tonometry → Eye rocky hard
- Optic Disc → Congestion at disc

Management 00:14:17

- Manage quickly as ganglion cells are dying
  i. Mannitol (Fast) – reduce the IOP
    - IV – 20% → 1-2mg/kg (250ml)

ii. Syrup glycerol
  - Non diabetic
  - 1-2ml/kg body wt
  - 6 table spoons

iii. Tab acetazolamide – Carbonic anhydrase inhibitor
  500mg → 2 stat
  - DOC for acute congestive glaucoma – Mannitol

iv. Pilocarpine → 2% → DOC → Causes Miosis

↓

Attack will be terminated

↓

Gonioscopy dove for further management on gonioscopy

↓

Angle closed + synechiae → If <180° peripheral iridotomy

↓

Laser iridotomy

↓

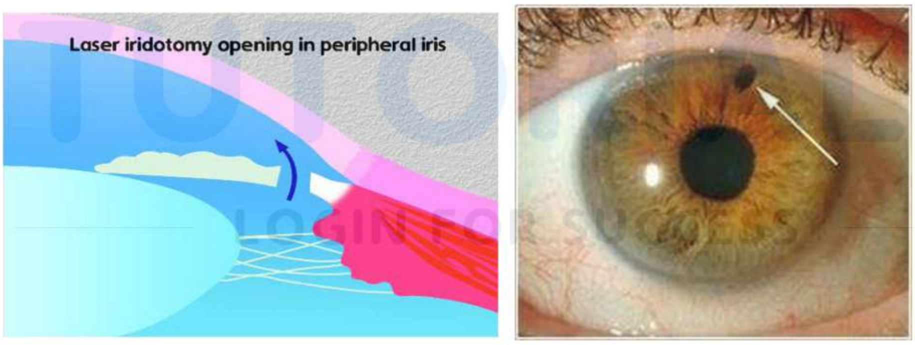


Fig: 4

↓

Peripheral iridotomy by ND Xay laser

↓

Opening dove – Mid peripheral part of iris

Mechanism – Photodisruption

Spurt size – 200mm generally at the crypts

↓

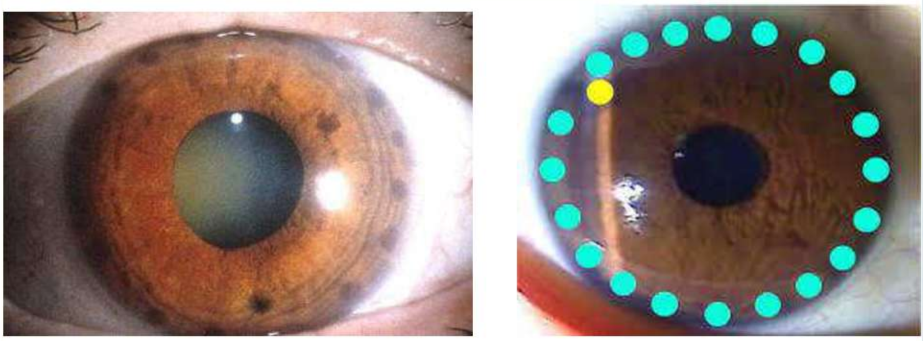


Fig: 5

↓

Peripheral laser rhinoplasty

↓

Constrict iris near collection channel

Acute angle closure glaucoma

- Vogt triad → Previous ACG attack
- P – Poor pupillary dilatation
- I – Iris atrophy
- G – Glaucomphlecan

Creeping angle closure

- Closure occurs more evenly in all quadrants, circumferential synechial closure so that the angle progressively becomes shallower.
- The appearance over times is of progressively more anterior iris insertion.

Plateau iris

- Plateau iris configuration
- Central ACD normal
- Convex iris from periphery

- Patients might have high IOP
- Patients might not get relief by peripheral iridotomy

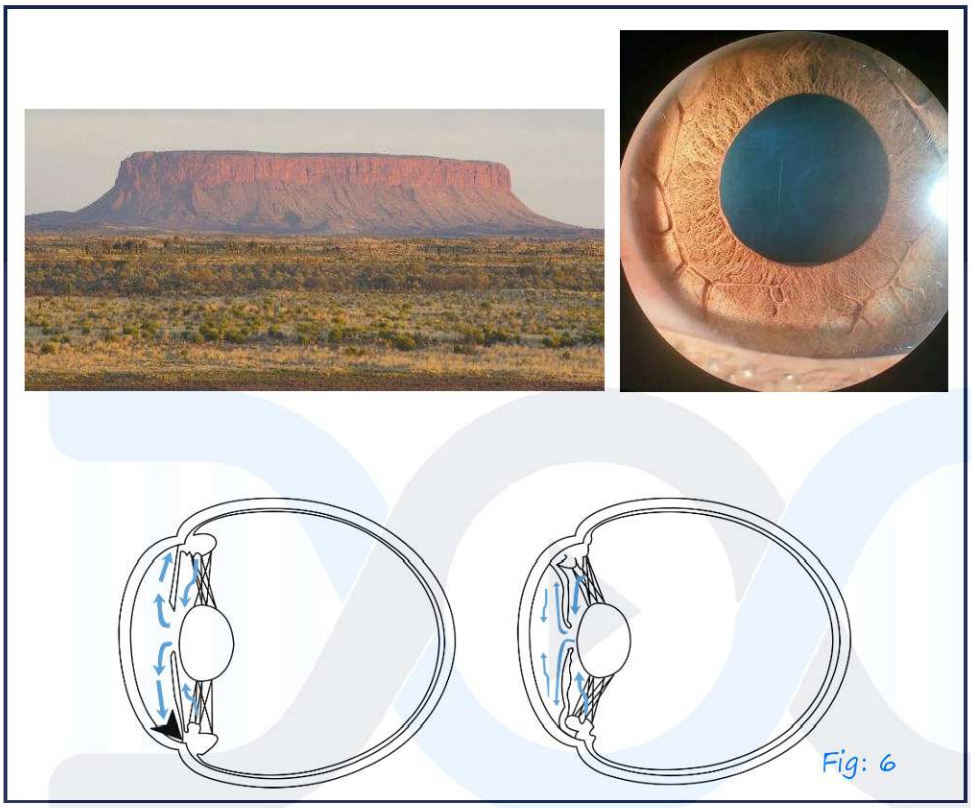


Fig: 6

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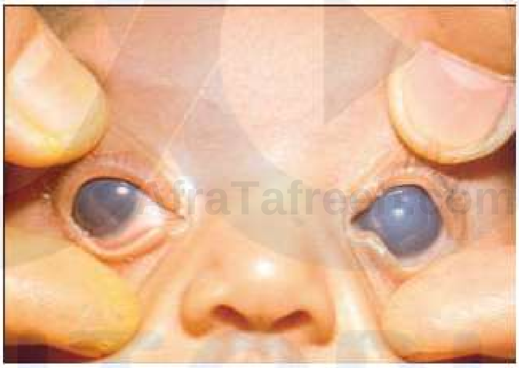
Signs: -

- Cornea appears large megalocornea
  - hazy
  - edema +
- Lens large, flat → Subluxation
- Eye ball large
- Sclera stretched
- Retina stretched
- Choroid stretched
- Maximum stretching's or lengthening occurs @limbus



Buphthalmos
↓
Bull's eye Fig: 1

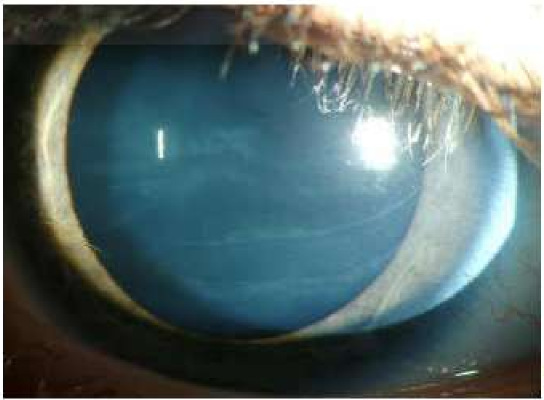


Fig: 2

TUTORIALS LOGIN TO SUCCESS

CONGENITAL GLAUCOMA

- Trabeculo dysgenesis (malformed)

Types 00:01:12

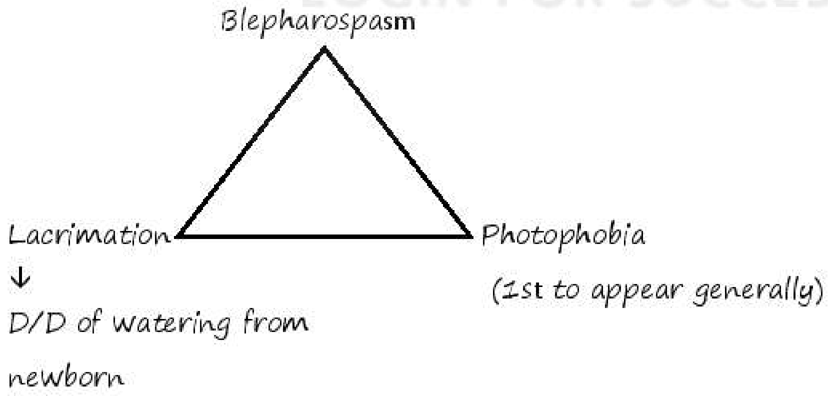
- True congenital: glaucoma present since birth
- Infantile: glaucoma appears after birth and within 3 years
- Juvenile: After 3 years-16 years
- Most Common type is Infantile Glaucoma

Etiology 00:02:29

- Can be due to MYOC/CYP gene mutation
- 1:10000 live births

Clinical features 00:02:56

- Classical triad


Blepharospasm
Lacrimation
↓
D/D of watering from newborn
Photophobia
(1st to appear generally)

- Haab's striae → Horizontal stress lines present at level of Descements membrane
  → Generally, they persist even after IOP is control

Loch ness monster Phenomenon 00:08:54

Fig: 3

- Vessel from major arterial circle project from the angle over root of iris (like aquatic bird of Scotland)
- Barkan's membrane
- Gonioscopic findings

Management 00:10:43

- Examine → Under anesthesia
  ↓
  - Measure corneal diameter
    ↓
    White to white limbal horizontal diameter
  - Gonioscopy
  - Measure IOP
  - Disc changes → cupping (reversible)

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- Surgery → definitive treatment of choice

Goniotomy

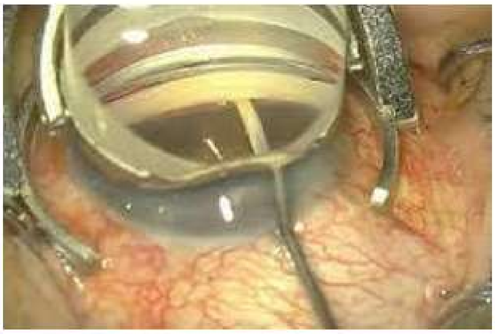

Fig: 4

- Place Balkan knife under gonioscopic new and cut open trabecular meshwork
- Only helpful and done only when clear corneal is present

Trabeculotomy

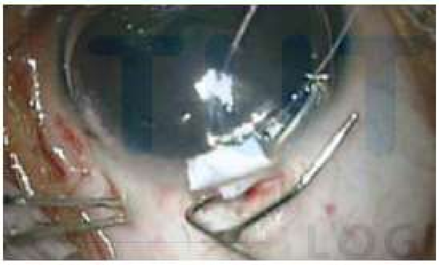

Fig: 5

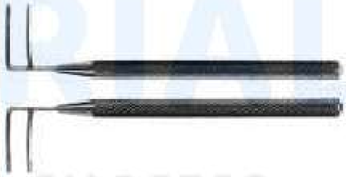

Fig: 6

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- Trabeculotomy → 2 drops for each eye

  ↓

  → Outer one is guarding and inner one is going into the meshwork

Trabeculotomy + Trabeculectomy

- Treatment of choice

SECONDARY GLAUCOMA

Pseudo exfoliation glaucoma 00:00:46

- Most common 2° OAG
- Pseudo exfoliative material is released from capsule of lens
- Mostly found in Scandinavian countries

Clinical features

- Knocks – Pseudo exfoliative material is on pupillary margin
- Zonules - becomes weak
- Lens subluxation → nuclear cataract
- Poor pupillary dilation
- Cornea – Krukenberg spindle
- Bull's eye sign/Target sign

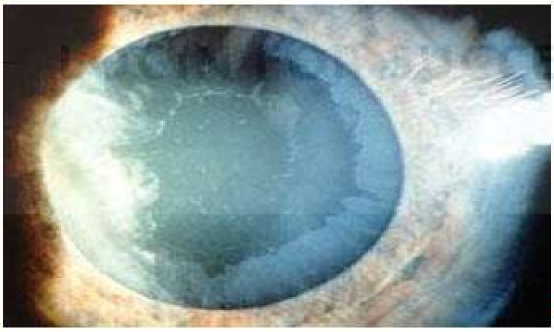


Fig: 1

- D/T movement of iris
- Seen on anterior lens capsule

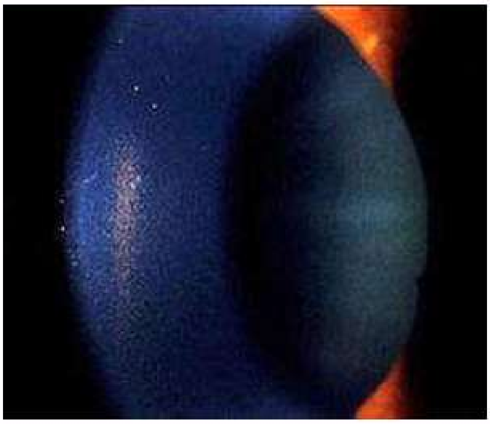


Fig: 2

- Also, most 90% cases turn into 2° open angle glaucoma

Treatment

- PG analogues
- Laser
- Trabeculectomy

Pigmentary glaucoma 00:07:11

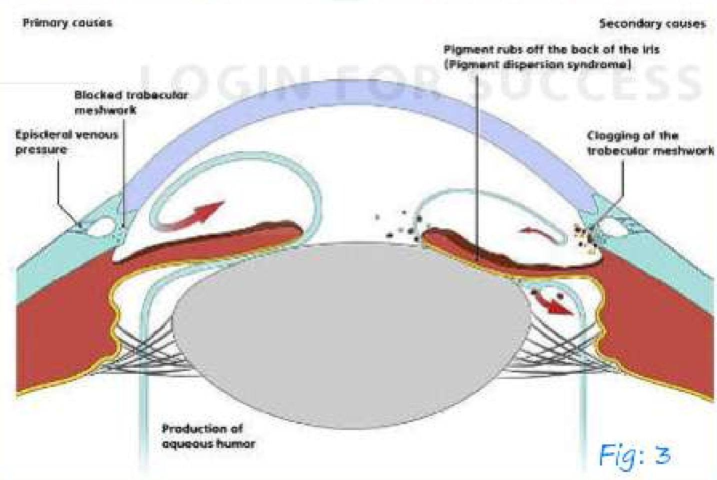
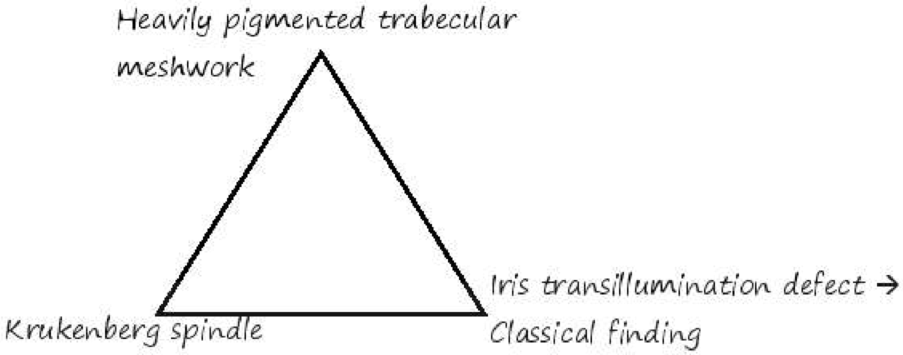

Primary causes
Secondary causes
Pigment rubs off the back of the iris (Pigment dispersion syndrome)
Blocked trabecular meshwork
Episcleral venous pressure
Clogging of the trabecular meshwork
Production of aqueous humor

Fig: 3

- Seen in young/males/myopes, when they exercise, concave iris configuration

- Reverse pupillary block → Higher pressure in anterior chamber
- Clinical features: Triad


Heavily pigmented trabecular meshwork
Krukenberg spindle
Iris transillumination defect → Classical finding

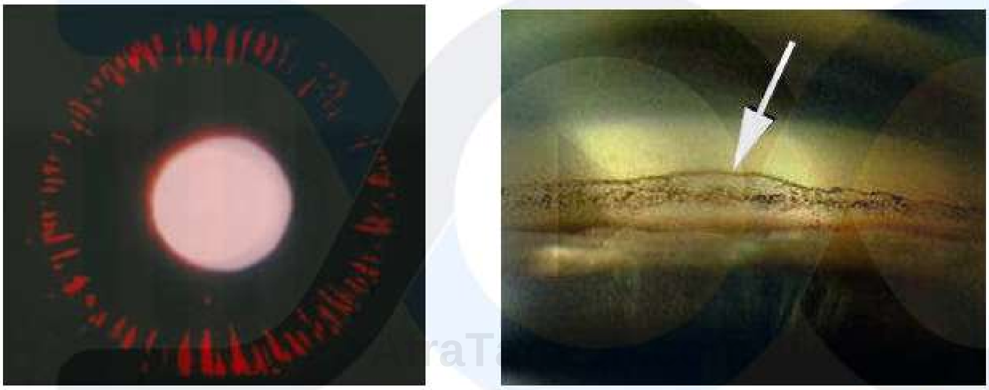


Fig: 4

- Sampoaeli's line → Heavily pigmented Schwabe's line on gonioscopy

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Neovascular glaucoma 00:12:55

- 100day glaucoma/Ischemia CRVO

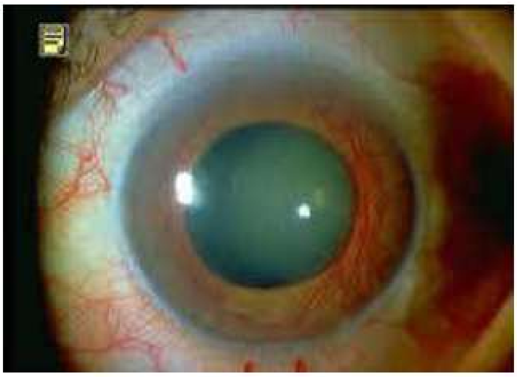

Fig: 5

- Causes
  - Ischemic CRVO
  - DR (Diabetic retinopathy)
  - BRVO
  - CRVO
  - Radiation
  - Tumor
  - Eale's disease
  - ROP
  - Leads to early 2° OAG
  - Later 2° ACG → because of fibrotic element
  - When there is vessel on iris, then its indication for PRP (Pan retinal photocoagulation)
  - Trabeculectomy

Malignant glaucoma 00:15:45

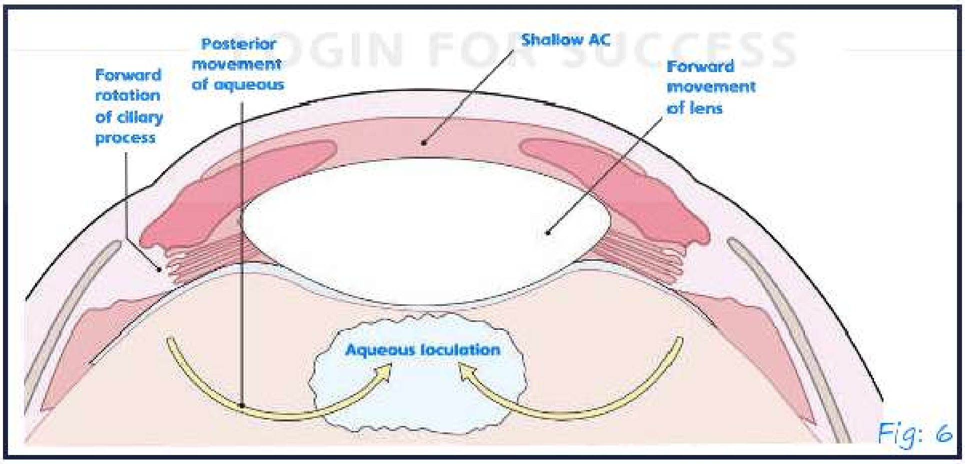
Posterior movement of aqueous
Shallow AC
Forward rotation of ciliary process
Forward movement of lens
Aqueous loculation

Fig: 6

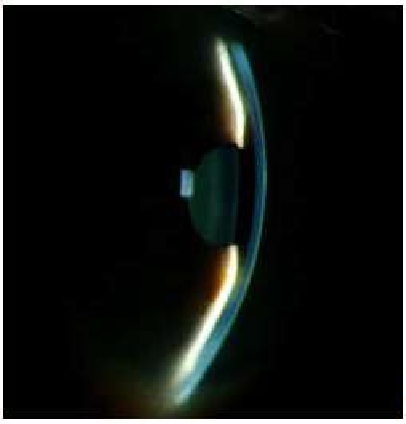

Fig: 7

- Cause:
- After any ocular surgery & laser
- Rarely after trauma
- When you do surgery ciliary process rotates arteria and touches lens → ciliolenticular block
- May touch vitreous – Ciliovitrial block

↓

Pushes aqueous humor in posterior segment

↓

Vitreous volume ↑

↓

Pushes lens

↓

Shallow anterior chamber

- Also called as 'aqueous misdirection syndrome'
- Most commonly occurs after trabeculectomy
- Example:
  - Atropine ointment → Push lens behind
  - Sclerotomy
  - Hyaloidotomy
  - PPV

Traumatic glaucoma 00:20:59

- Early hyphema specially 8 ball → Accumulation of RBC in angle
- Ghost cell glaucoma – Khaki closed RBC (following vitreous hemorrhage)
- Angle recession glaucoma → Occurs after years of injury

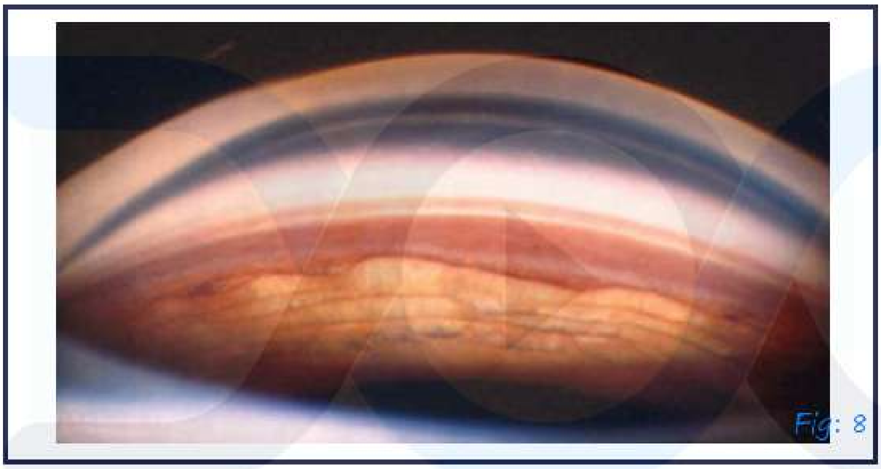

Fig: 8

Steroid induced glaucoma 00:23:16

Potency	Steroid	Glaucoma risk
High	Betamethasone Clobetasol propionate Dexamethasone Flucinonide	↑
Medium	Triamcinolone acetonide Loteprendole etabonate Dexamethasone sodium phosphate Fluormethalone	
Low	Hydrocortisone Rimexolone Medrisone	

- IOP rise by steroids
- Low - <6mmHg
- Intermediate – 6-15mmHg
- High >15mmHg

Mechanism

- Steroids stabilized lysosomal membrane → decrease their metabolism → Increased accumulation
- Reduced activity of matrix metalloproteinases accumulation of fibronetic elastic and type IV collagen
- Suppresses phagocytic activity of cells lining TM receptors may act on them to after cell migration and phago cytosis

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ICE syndrome 00:25:38

- 2° glaucoma where there is problem in endothelium of cornea
- ICE – Irido corneal endothelial syndrome
- Silver beaten/Metal appearance
- 30-50 age, females
- I-Iris nevus syndrome → Max glaucoma
- C – Chandler syndrome → Max corneal edema
- E – Essential iris atrophy
- Core atropia
- Iris atrophy

Axenfeld anomaly

- Axenfeld anomaly – Only posterior embryotoxon

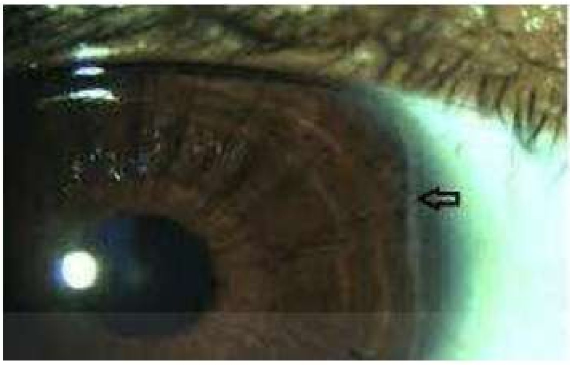


Fig: 9

- Rieger anomaly – posterior embryotoxon + corectopia, iris atrophy + extensive PAS, sclerocornea, excessive periumbilical skin, facial and dental anomalies

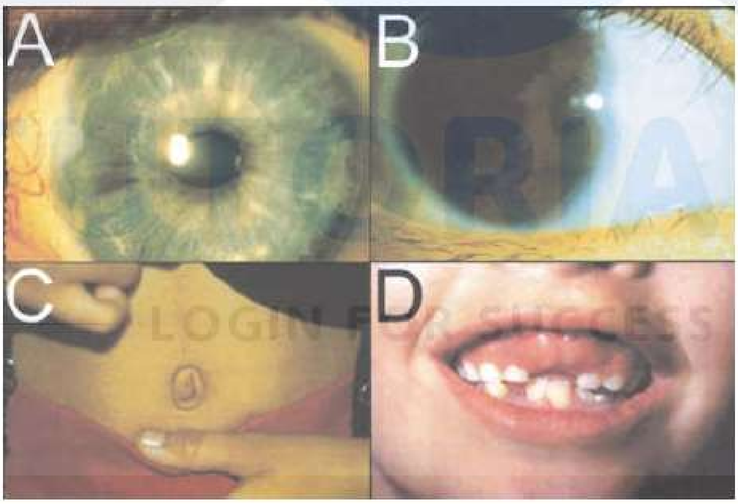
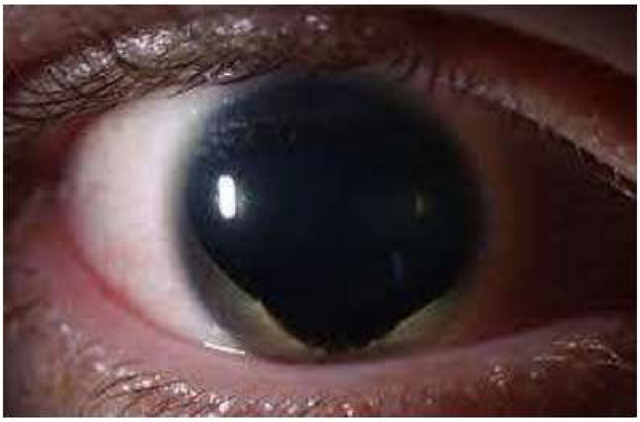


Fig: 10



Aniridia Fig: 11

Aniridia 00:30:02

- Some part of iris present most of iris tissue absent
- Congenital condition
- Mutation in PAX6/PITX2 gene
- Autosomal dominant – most common
- Most common associated with wilms tumor or gillepie syndrome
- Association with in
  - 2° glaucoma
  - Cataract
  - Nystagmus
  - Poor vision
  - Limbal stem cell deficiency

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MANAGEMENT OF GLAUCOMA

AAO- guidelines – Target IOP

	Clinical conditions	Target IOP
1	Glaucoma patients with mild damage (optic disc cupping but no VF loss)	Reduction of 20-30% from baseline
2	Glaucoma patients with advance damage	Reduction of 40% or more from base line
3	Normal pressure glaucoma or NTG	Reduction of 30% from baseline
4	Ocular hypertension	Reduction of 20% from base line
5	Open angle glaucoma with IOP in the mid to high 20a	Target IOP range 14-18 mmHg
6	Advanced glaucoma	Target IOP < 15 mmHg
7	OHT whose IOP > 30 mmHg with no sing of optic nerve damage	Target IOP < 20 mmHg

DOC TUTORIALS

Antiglaucoma drugs 00:02:00

Prostaglandin analogues

- Best drugs → max reduction of IOP
  ↓
  reduce 40% from base line
- Best drugs for – NTG
  - POAG
  - Ocular HTN
- MOA: increase uveoscleral outflow by relaxation of ciliary muscle ;
- Drugs: - Latanoprost → DOC (0.05%)
  - Travoprost (0.04%)
  - Bimatoprost → prostamide (0.03% - 0.01%)
  - Tafluprost
  - Unoprost
- Bimatoporst→ dual action → decreases production also

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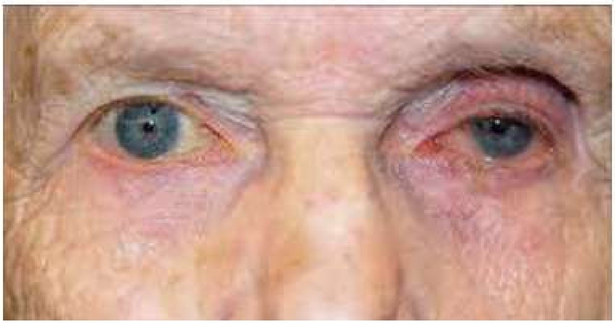
S/E of Prostaglandin analogues

- Redness
- Itching
- Hypertrichosis → FDA of treatment of Alopecia/madarosis
- Periocular pigmentation
- Iris pigmentation +
- Cystoid macular edema
- Reactivation of Herpes simplex keratitis



PAP

↓

Most common seen in bimatoprost

Fig: 1

- Inflammatory fibrotic changes to the eyelid or to Mueller's muscle, atrophy of existing adipocytes, and inhibition of adipogenesis
  - Upper lid ptosis
  - Deepening of the upper lid sulcus
  - Involution of dermatochalasis
  - Periorbital fat atrophy
  - Mild enophthalmos
  - Inferior scleral show
  - Increased prominence of lid vessels
  - Tight eyelids

β Blockers

- Timolol (Nonselective)
- Betoxolol (β selective) (pulmonary safe)
- Decreases production of aqueous (inhibit C-AMP)
- S/E: - A- Allergic Blepharo conjunctivitis
  - B- Bradycardia, Bronchospasm

  - C- Cardiac conduction defects(arrhythmias)
  - D- Dry eye
- C/I: - Heart Disease
  - Asthma
  - Normal tension glaucoma because of Nocturnal HTN

α-Agonists 00:13:18

- Non- selective → epinepheline
  → depivefrine
- Selective → Brimomidine - alpha 2 selective
  → Apraclonidine → α_2 selective + α_1 weak selective action
- S/E: - redness, itching
  - Apraclonidine → lid retraction
    → mydriasis
    → Tachyphylaxis
  - Brimonidine → Sedation - specially children
- C/I in young children as it crosses blood brain barrier
- Epinephrine → CMF in aphakia
  → Adreno chrome deposit
- Decrease Production + increase uveoscleral outflow (IOP reduction in 20-25 % from base line)

Carbonic anhydrase inhibitor 00:17:16

- MOA : Decreases production of aqueous by inhibiting carbonic anhydrase
- Acetazolamide - only oral form (250 mg)
- Dorzolamide -2% gel
- Brinzolamide
- S/E: - Gastritis- most common
    - Bitter taste in mouth
    - Tingling over mouth,fingers
    - Cardiac overload
    - Metabolic acidosis
    - Renal stones
- C/I: - Sulpha allergy
    - Careful to be given in sickle cell trait anomaly

Cholinergic drugs 00:19:56

- Eg. pilocarpine
- MOA :1. Increase TM outflow
- 2.Miosis → angle closure glaucoma
- S/E- Transient myopia → brow ache
    - Anterior cataract
    - Retinal detachment
    - Punctual stenosis
    - Inflammation

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Newer antiglaucoma agents 00:22:07

Latanoprostene bunod (LBN)

- LBN is a single entity NO-donating prostaglandin F2α analogue. Rapidly metabolized by esterases into latanoprost acid and butanediol mononitrate (BDMN), an NO- donating moiety. Nitric Oxide increase conventional outflow via the trabecular meshwork and Schlemm's canal.

ROCK-Inhibitors

- Like nitric oxide, work primarily by:
- 1. enhancing trabecular meshwork outflow.
- 2. Inhibition of the norepinephrine transporter system, which leads to decreased production AH
- 3.Lowering of episcleral venous pressure
- Rock inhibitor – Nitarsudil
  - Rho kinase inhibitor
- S/E of Rock inhibit → whorl/vortex keratopathy

Rocklation

–Combination of Latanoprost /Netarsudil

↓

Now available in fixed combination with latanoprost

Lasers in glaucoma 00:24:53

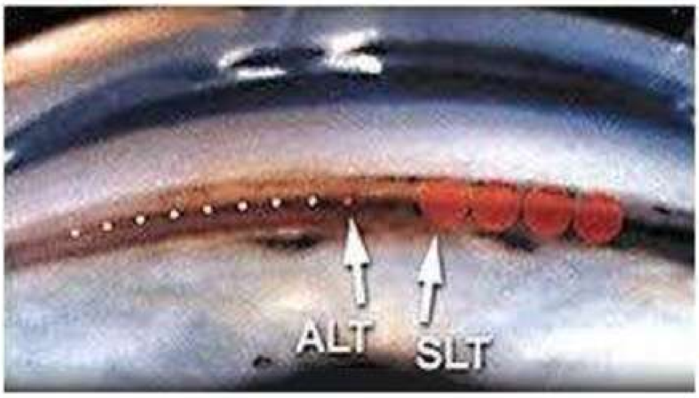

Fig: 2

- ALT- Argon laser trabeculoplasty
- SLT- Selective laser trabeculoplasty

ALT

- On TM
- Smaller size
- Generates heat and damages TM

  ↓

  Shrinkage of some of TM
- Not much preferred because it causes heat related damage

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SLT

- Done by double frequency Nd YAG laser
- Biochemical change
- No damage or minimal damage to surrounding structures

Trabeculectomy

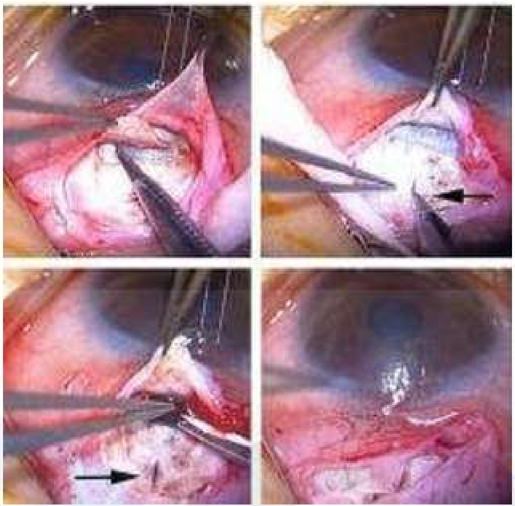


Fig: 3

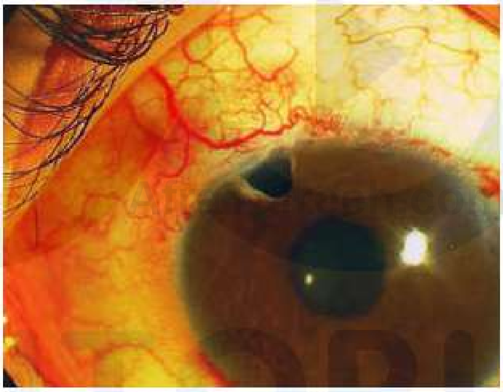


Fig: 4

- First, a conjunctival flap is created — Limbal based / Fornix bases

↓

Partial thickness scleral flap created

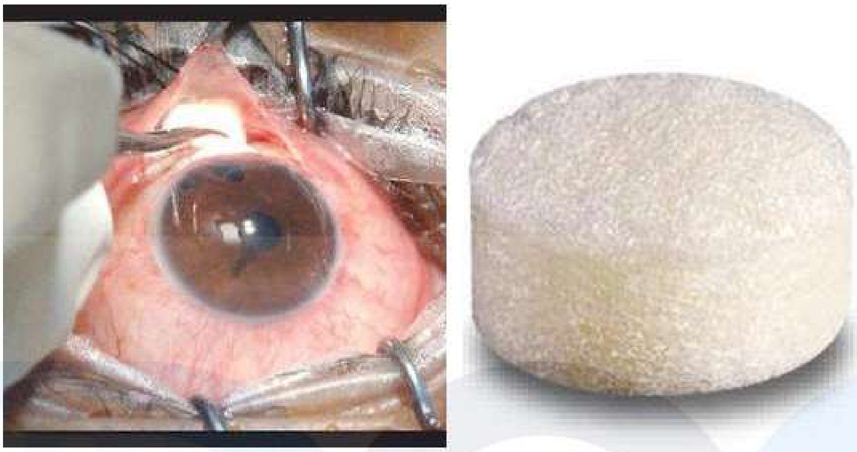
↓

Cut part of TM and peripheral iridotomy

- Fistula is created for drainage of aqueous humor directly to subconjunctival space

To decreases fibrosis of fistula

- Mitomycin C > 5-Fluoro Uracil
- Anti VEGF's



Ologen

Fig: 5

- Biodegradable

  ↓

  Collagen based product
- Self-degraded in 90-180 days

Shunts /Valves

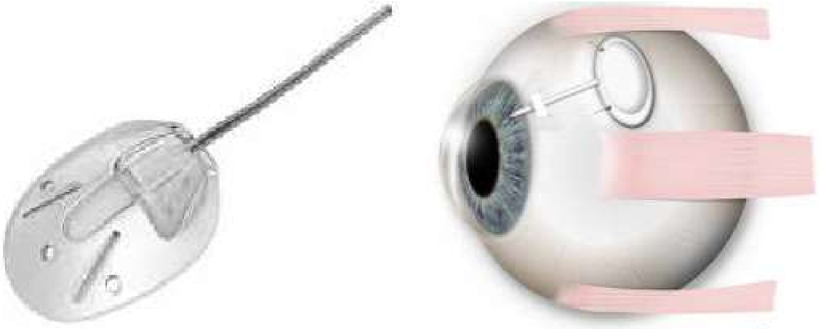


Fig: 6

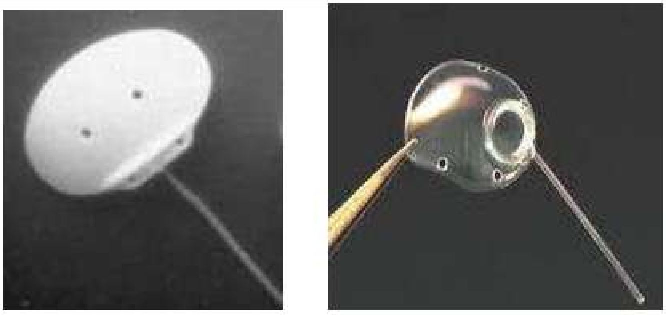


Fig: 7

- Valves → 2 ends
  - Flat end → subconjunctival space
  - Tube end → anterior chamber

→ Implants
  - Valved
    - ↓
    - Ahmed
    - Kruplin
  - Non valved
    - ↓
    - Molteno
    - Baer veldt

- Indications → Neovascular glaucoma
- For → post VR surgery
- Trabeculitis → Post PK surgeries
- With valve → Inflammatory
- Shunts → ICE

Minimally invasive glaucoma surgery 00:36:42

- Safe, control IOP
- Minimal manipulation of ocular anatomy

Express Mini Shunt Fig: 8

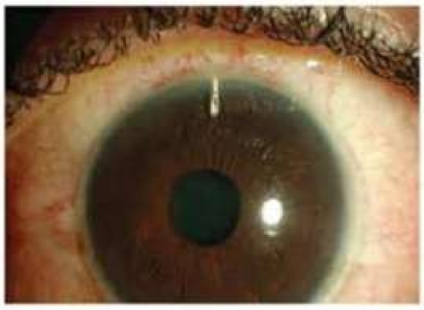


Fig: 9

Express mini shunt

- 3mm stainless steel
- 400-micron external diameter
- 50-micron lumen
- MRI compatible
- 26 G needle under conjunctival and scleral flap
- Shunt is placed

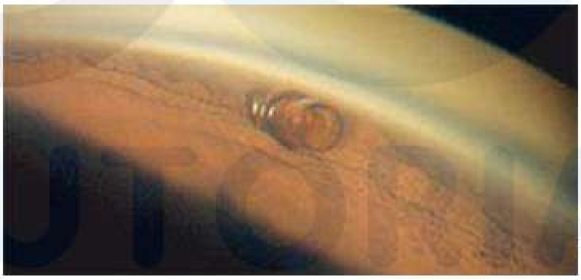


Fig: 10

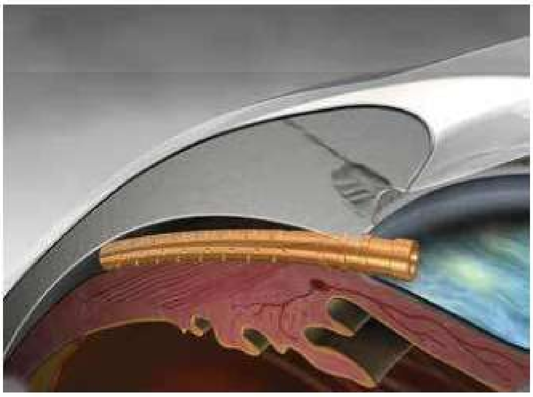


Fig: 11

Cypass micro shunt

- Creates a cyclo dialysis cleft
- Permanent conduit from AC to the suprachoroidal space
- Drains aqueous internally rather than accessing Schlemm's canal.
- Implanted into the supraciliary space
- Cypass micro shunt → cyclodialysis cleft created

Hydrus micro shunt Fig: 12

Made up of Nitinol(nickel + titanium)

Implanted into Schlemm's canal, occupying 90 degrees of angle at the time of cataract surgery

Very much like a coronary Stent ,it keeps Schlemm's canal open and prevents it from collapsing

Trabectome Fig: 13

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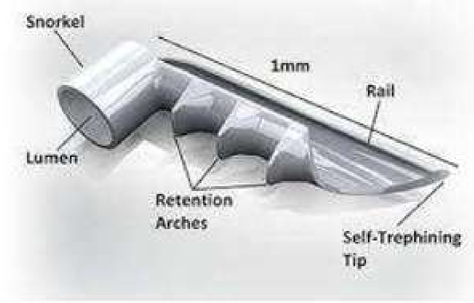
Snorkel
1mm
Rail
Lumen
Retention Arches
Self-Trephining Tip

I stent

Fig: 14

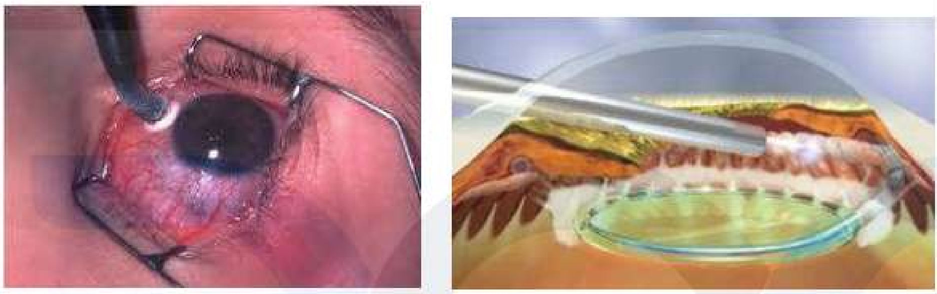

Fig: 15

TUTORIALS
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Cyclodestructive procedure 00:42:00

↓

- Intractable glaucoma
    - Failed trabeculectomy Sx
    - No option to control IOP
    - Blind painful eye
- Done on sclera → Ischemic necrosis ciliary process at sclera

  ↓

  Intracellular crystals formed
- Done by lasers → Endoscopic laser diode